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Sam'l Lewis M.D.

TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME XIII.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM SEPTEMBER, 1885, TO JULY, 1887.

EDITED BY

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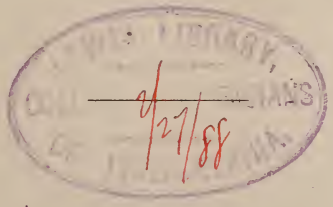
MEDICAL REGISTRAR OF THE UNIVERSITY HOSPITAL; PHYSICIAN TO THE OUT-PATIENT DEPARTMENT OF THE CHILDREN'S HOSPITAL; RECORDER OF THE SOCIETY.



PHILADELPHIA:

PRINTED FOR THE SOCIETY BY WM. J. DORNAN.

1887.

THE present volume includes the Transactions of the Society from September, 1885, to June, 1887, inclusive.

The papers by Dr. Warren, on A Comparison of the Changes in Arteries after Ligature, and in the Ductus Arteriosus and Umbilical Arteries after Birth (p. 75); by Dr. Piersol, on Medical Photography (p. 243); by Dr. Osler, on The Hæmatozoa of Malaria (p. 255); and by Dr. Welch, on The Structure of White Thrombi (p. 281), were read by the respective authors at the semi-annual conversational meetings of the Society, they having been appointed to open the proceedings on those evenings.

In the Sections devoted to "Miscellaneous" are placed several papers which arrived too late to be inserted in the Sections to which they properly belong.

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FORMER PRESIDENTS.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., elected 1857.

RENÉ LA ROCHE, M.D., elected 1858.

ALFRED STILLÉ, M.D., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M.D., elected 1860 and '63.

J. M. DA COSTA, M.D., elected 1864, '65, and '66.

JOHN H. PACKARD, M.D., elected 1867 and '68.

S. WEIR MITCHELL, M.D., elected 1869.

JOHN ASHHURST, Jr., M.D., elected 1870.

JAMES H. HUTCHINSON, M.D., elected 1871 and '72.

WILLIAM PEPPER, M.D., elected 1873.

H. LENOX HODGE, M.D., elected 1876.

S. W. GROSS, M.D., elected 1879.

JAMES TYSON, M.D., elected 1882 and '83.

E. O. SHAKESPEARE, M.D., elected 1884.

OFFICERS AND COMMITTEES

OF THE

PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the annual meeting, October, 1886.)

President.

J. C. WILSON, M.D.

Vice-Presidents.

J. H. C. SIMES, M.D.,
F. P. HENRY, M.D.,
J. M. BARTON, M.D.,
H. F. FORMAD, M.D.

Secretary.

J. K. MITCHELL, M.D.

Recorder.

W. E. HUGHES, M.D.

Treasurer.

M. S. FRENCH, M.D.

Curator.

H. F. FORMAD, M.D.

Committee on Publication.

J. H. MUSSER, M.D., HENRY BEATES, JR., M.D.,
H. R. WHARTON, M.D., F. P. HENRY, M.D.,
W. E. HUGHES, M.D. (ex-officio).

Committee on Morbid Growths.

G. E. DE SCHWEINITZ, M.D., MORRIS LONGSTRETH, M.D.,
WILLIAM OSLER, M.D., B. A. RANDALL, M.D.,
F. P. HENRY, M.D. GUY HINSDALE, M.D.

Business Committee.

W. A. EDWARDS, M.D., HENRY BEATES, JR., M.D.,
J. H. MUSSER, M.D., JAMES TYSON, M.D.,
B. A. RANDALL, M.D.

LIST OF MEMBERS.

Members marked O. M. are those originating the Society.
Members marked N. R. are non-resident.

ELECTED

1857 AGNEW, D. HAYES, Rhea Barton Professor of Surgery in the University of Pennsylvania, 1601 Walnut Street. (O. M.)
1871 ALISON, ROBERT H., resigned 1884.
1864 ALLEN, HARRISON, resigned December 11, 1879.
1869 ALLIS, OSCAR H., resigned February 24, 1881.
1887 ALLYN, HERMAN B., 737 North Forty-first Street.
1865 ANDREWS, THOMAS H., forfeited membership by non-payment of dues.
1871 ASHBRIDGE, WILLIAM, resigned December 11, 1879.
1867 ASHHURST, FRANCIS, Mount Holly, New Jersey. (N. R.)
1861 ASHHURST, JOHN, JR., Professor of Clinical Surgery in the University of Pennsylvania, 2000 De Lancey Place.
1863 ASHHURST, SAMUEL, resigned December 22, 1887.
1871 ATLEE, WASHINGTON L., JR., forfeited membership by non-payment of dues, December 10, 1874.

1866 BACHE, T. HEWSON, resigned December 27, 1883.
1878 BAER, B. F., resigned.
1887 BAKER, G. F., 1818 Spruce Street.
1876 BARR, J. W., forfeited membership by non-payment of dues, December 8, 1881.
1880 BARTHOLOW, ROBERTS, Professor of Materia Medica in the Jefferson Medical College, 1525 Locust Street.
1868 BARTLES, WM. H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)
1883 BARTON, ISAAC, 128 South Fifteenth Street.
1869 BARTON, J. M., Surgeon to the German and Jefferson Medical College Hospitals, 1344 Spruce Street.
1885 BEATES, HENRY, JR., 1421 Girard Avenue.
1873 BEECHER, A. C. W., 523 South Ninth Street.
1870 BELL, J. R. F., forfeited membership by non-payment of dues, December 10, 1874.
1871 BENNETT, WM. H., 332 South Fifteenth Street.
1863 BENTON, C. H. (N. R.)
1887 BERENS, BERNARD, 2006 Arch Street.

ELECTED

1875 Bernardy, Eugene P., resigned September 22, 1881.
1870 Bertholet, Robert Morris, died 1882.
1871 Betts, Thomas, Branchtown, Pa. (N. R.)
1860 Bishop, C. S., died.
1866 Black, J. J., New Castle, Delaware. (N. R.)
1863 Boardman, C. H., St. Paul, Minnesota. (N. R.)
1886 Bodamer, George A., German Hospital.
1880 Bœnning, Henry C., forfeited membership by non-payment of dues, April 14, 1882.
1857 Boker, Charles S., Surgeon to St. Joseph's Hospital, 212 South Fifteenth Street. (O. M.)
1868 Bolles, Lucius S., died August 15, 1873.
1883 Boyd, George M., 1743 North Seventeenth Street.
1877 Bradford, T. Hewson, resigned December 23, 1880.
1873 Bray, Daniel, forfeited membership by non-payment of dues, December 8, 1881.
1883 Breinig, P. B., Bethlehem, Pennsylvania.
1857 Brinton, John H., Professor of the Practice of Surgery and Clinical Surgery in the Jefferson Medical College, 1423 Spruce Street. (O. M.)
1874 Brubaker, A. P., 1210 Race Street.
1874 Bruen, E. T., Professor of Physical Diagnosis in the University of Pennsylvania, Physician to the Philadelphia Hospital, 1814 Locust Street.
1871 Buck, William Penn, resigned October 23, 1873.
1867 Burnett, Charles H., resigned December 8, 1881.

1882 Cathcart, James H., died March 5, 1874.
1879 Cathcart, T. H., died March 28, 1882.
1887 Cattell, H. W., 222 South Thirty-ninth Street.
1887 Chambers, G. H., 739 North Seventeenth Street.
1871 Cheston, C. Morris, West River, Maryland. (N. R.)
1865 Cheston, D. Murray, resigned December 13, 1883.
1887 Chrystie, Walter, 125 South Sixteenth Street.
1871 Clark, Leonardo S., resigned November 29, 1883.
1866 Cleemann, Richard A., Physician to St. Mary's Hospital, 2135 Spruce Street.
1872 Cohen, J. Solis, Professor of Diseases of the Throat and Chest in the Philadelphia Polyclinic and School for Graduates, 1431 Walnut Street.
1883 Cohen, S. Solis, 219 South Seventeenth Street.
1887 Collins, James, 704 Franklin Street.
1869 Corbit, William B., Washington, D. C. (N. R.)
1877 Cruice, R. B., resigned December 22, 1887.
1871 Curtin, Roland G., Physician to the Philadelphia Hospital, 22 South Eighteenth Street.

ELECTED

1857 Da Costa, J. M., Professor of the Theory and Practice of Medicine in the Jefferson Medical College, 1700 Walnut Street. (O. M.)
1885 Daland, Judson, 319 South Eighteenth Street.
1859 Darby, J. T., died.
1876 Darlington, W. L. (N. R.)
1857 Darrach, James, 5094 Green Street, Germantown. (O. M.)
1868 Darrach, William, died January 28, 1881.
1887 Davis, E. P., resigned.
1882 Davis, G. G., 1817 Mount Vernon Street.
1885 Deaver, J. B., Demonstrator of Anatomy in the University of Pennsylvania, Surgeon to the Philadelphia Hospital, and to the German Hospital, 1610 Vine Street.
1877 Deaver, R. W., resigned January 27, 1887
1882 Dercum, F. X., Pathologist to the State Hospital for the Insane at Norristown, Pennsylvania; Chief of Dispensary for Nervous Diseases, University Hospital, Neurologist to the Philadelphia Hospital, 636 North Eighth Street.
1882 De Schweinitz, G. E., Ophthalmic Surgeon to the Children's Hospital, and to the Philadelphia Hospital, 1330 Spruce Street.
1885 Dock, George, 110 South Eighteenth Street.
1886 Donges, John W., Camden, New Jersey.
1877 Drysdale, T. M., 1531 Arch Street.
1883 Dunn, T. D., West Chester, Pennsylvania. (N. R.)
1866 Duer, Edward L., 1704 Arch Street.
1870 Duhring, L. A., resigned November 9, 1882.
1876 Dulles, C. W., 4101 Walnut Street.
1860 Dunton, W. R., 5059 Germantown Avenue, Germantown.
1883 Dunmire, G. B., 1116 Arch Street.
1863 Dyer, Ezra, Pittsburg, Pa. (N. R.)

1883 Edwards, J. F., forfeited membership by non-payment of dues.
1882 Edwards, W. A., Physician to St. Joseph's Hospital, 300 South Thirteenth Street.
1885 Egbert, J. C., General Wayne, Pennsylvania.
1875 Engel, Hugo, forfeited membership by non-payment of dues, February 23, 1882.
1876 Eskridge, J. T., Colorado Springs, Colorado. (N. R.)

1881 Fenton, T. H., 1335 Arch Street.
1857 Fischer, Emil, 729 North Sixth Street. (O. M.)
1863 Fish, Augustine H., died August 3, 1872.
1876 Fisher, Henry M., Physician to the Episcopal Hospital, 919 Walnut Street.
1857 Forbes, William S., forfeited membership by non payment of dues.
1869 Ford, William H., 1622 Summer Street.

ELECTED

1878 Formad, H. F., Demonstrator of Pathology in the University of Pennsylvania, 3535 Locust Street.
1871 Fox, Charles, resigned December 10, 1884.
1886 Freeman, W. J., 2034 Chestnut Street.
1880 French, M. S., 1437 Spruce Street.
1885 Fussell, M. H., 168 Green Lane, Manayunk.

1883 Garrett, E. F., 5043 Germantown Avenue, Germantown.
1868 Garretson, James E., resigned November 22, 1883.
1871 Gerhard, George S., Ardmore, Pennsylvania. (N. R.)
1870 Getchell, Frank H., 1432 Spruce Street.
1877 Gilliard, Louis, died November, 1878.
1882 Godey, H. E., N. E. corner Nineteenth and Spruce Streets.
1877 Goldsborough, C. B. (N. R.)
1866 Goodell, William, Clinical Professor of Diseases of Women in the University of Pennsylvania, 1418 Spruce Street.
1886 Goodell, W. C., 1418 Spruce Street.
1868 Goodman, H. Ernest, resigned February 23, 1882.
1868 Graham, James, resigned January 9, 1879.
1867 Grier, Matthew J., 1531 Spruce Street.
1883 Griffith, J. P. C., Assistant Physician to the University Hospital, 123 South Eighteenth Street.
1868 Gross, Ferdinand H., Surgeon to the German Hospital, 1416 Girard Avenue.
1885 Gross, Onan B., 700 Market Street, Camden, New Jersey.
1857 Gross, Samuel D., died May 6, 1884. (O. M.)
1858 Gross, Samuel W., Professor of the Principles of Surgery and Clinical Surgery in the Jefferson Medical College, 1112 Walnut Street.
1869 Grove, John H., Surgeon to St. Mary's Hospital, 1330 Arch Street.
1862 Gruel, Theodore H. E., 612 Spruce Street.
1874 Guitéras, John, Charleston, South Carolina. (N. R.)

1884 Haehlen, W. F., 1421 North Broad Street.
1871 Hale, George J., forfeited membership by non-payment of dues, March 23, 1876.
1857 Hall, A. Douglass, resigned October 9, 1873. (O. M.)
1886 Hamaker, W. D., Meadeville, Pennsylvania. (N. R.)
1875 Hand, Frank C., died September 1, 1881.
1885 Hare, Hobart A., 117 South Twenty-second Street.
1866 Hare, Horace Binney, died March 21, 1879.
1868 Hargadine, R. W. (N. R.)
1859 Harlan, George C., resigned January 12, 1882.
1874 Harris, Charles M., died.
1858 Harris, Robert P., resigned January 9, 1873.
1881 Hart, R. H., forfeited membership by non-payment of dues.

ELECTED

1857 Hartshorne, Edward, died June 29, 1885. (O. M.)
1859 Hartshorne, Henry, resigned October 12, 1864.
1867 Hassler, Ferdinand A., resigned February 9, 1871.
1869 Hatfield, Nathan L., died.
1868 Hays, I. Minis, resigned January 13, 1881.
1879 Hazlehurst, S. F., Colorado Springs, Col. (N. R.)
1873 Hearn, Joseph, Surgeon to the Jefferson Medical College Hospital, Surgeon to the Philadelphia Hospital, 1120 Walnut Street.
1870 Henry, Frederick P., Physician to the Episcopal Hospital, and to the Philadelphia Hospital, 721 Pine Street.
1866 Herbert, Theodore. (N. R.)
1879 Hess, Robert, resigned January 12, 1882.
1857 Hewson, Addinell, forfeited membership by non-payment of dues, December 22, 1881. (O. M.)
1880 Hewson, Addinell, Jr., 332 South Sixteenth Street.
1874 Hickman, N. B., forfeited membership by non-payment of dues, December 8, 1881.
1884 Hinsdale, Guy, 4004 Chestnut Street.
1858 Hodge, H. Lenox, died June 10, 1881.
1883 Hoelly, G. H., forfeited membership by non-payment of dues.
1871 Hoffman, Washington Atlee, died September 20, 1874.
1886 Holland, J. W., Professor of Chemistry in the Jefferson Medical College, 1914 Locust Street.
1860 Hopkins, Henry St. George. (N. R.)
1876 Hopkins, W. B., resigned October 14, 1880.
1877 Horwitz, Theodore, died December 12, 1877.
1870 Houston, James P. S., Savannah, Georgia. (N. R.)
1866 Howe, Herbert M., resigned February 28, 1878.
1859 Hoyt, William D. (N. R.)
1880 Hughes, D. E., Williamsport, Pennsylvania. (N. R.)
1882 Hughes, W. E., Medical Registrar of the University Hospital, 3726 Baring Street.
1879 Huidekoper, R. S., resigned October 28, 1880.
1857 Humphries, George H., New York City. (O. M., N. R.)
1868 Hunter, Charles T., resigned October 14, 1880.
1870 Hutchins, E. R. (N. R.)
1858 Hutchinson, James H., Physician to the Pennsylvania Hospital, 133 South Twenty-second Street.

1866 Ingham, James V., resigned December 11, 1879.
1866 Ingram, T. D., forfeited membership by non-payment of dues, December 8, 1881.

1874 Jamison, Edward, St. Louis, Missouri. (N. R.)
1866 Jenks, William, died October 31, 1881.

ELECTED

1875 Johnson, Russell H., resigned December 8, 1881.
1883 Judd, L. D., 3603 Powelton Avenue.
1885 Jurist, L. J., 1210 Walnut Street.

1859 Kane, John K., Wilmington, Delaware. (N. R.)
1857 Kealer, William, resigned January 28, 1875. (O. M.)
1874 Keating, John M., 1504 Walnut Street.
1857 Keating, William V., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)
1866 Keen, William W., resigned December 9, 1886.
1875 Kelly, Joseph V., 4257 Main Street, Manayunk.
1884 Kilduff, Robert, resigned December 9, 1886.
1858 King, William, died.
1879 Kirkbride, J. J., 35 South Nineteenth Street.
1869 Knight, Samuel R., resigned October 28, 1880.

1867 Lambdin, A. C., forfeited membership by non-payment of dues, December 10, 1874.
1871 Landis, Henry G., died.
1872 La Roche, C. Percy, Nice, France. (N. R.)
1857 La Roche, René, died December 9, 1872. (O. M.)
1879 Leach, A. L., forfeited membership by non-payment of dues, December 22, 1881.
1869 Leaman, Henry, 828 North Broad Street.
1860 Lee, Charles C., New York City. (N. R.)
1882 Lee, J. G., resigned September, 1883.
1861 Leedom, John M., died.
1887 Leopoldt, Isaac, 1520 Franklin Street.
1887 Leuf, A. H. P., 240 South Eighth Street.
1857 Levick, James J., resigned October 13, 1861. (O. M.)
1868 Lewis, Francis W., 2016 Spruce Street.
1869 Lewis, Fred. W., died 1873.
1885 Lewis, George T., 5059 Germantown Avenue.
1875 Lewis, Morris J., Physician to the Episcopal Hospital, 1216 Walnut Street.
1857 Lewis, Samuel, Aldine Hotel. (O. M.)
1877 Little, William S., 215 South Seventeenth Street.
1859 Livezy, Edward, forfeited membership by non-payment of dues, September 14, 1864.
1886 Lloyd, James Hendrie, Neurologist to the Philadelphia Hospital, 200 South Fortieth Street.
1876 Loder, P. E., forfeited membership by non-payment of dues, February 9, 1882.
1886 Longaker, D., 513 Green Street.

ELECTED

1873 Longenecker, Jerome, forfeited membership by non-payment of dues, 1875.

1875 Longstreth, Morris, Physician to the Pennsylvania Hospital, 1416 Spruce Street.

1870 Loughlin, J. Eneu, forfeited membership by non-payment of dues, May 9, 1878.

1882 MacConnell, W. G., forfeited membership by non-payment of dues.

1873 MacCoy, A. W., 1415 Walnut Street.

1874 Mann, Charles H. (N. R.)

1867 Markoe, James, died March, 1884.

1887 Martin, Edward, 415 South Fifteenth Street.

1868 Martin, George. (N. R.)

1874 Matteson, Charles C., died.

1865 Maury, Frank F., died 1879.

1870 Maxwell, J. G., forfeited membership by non-payment of dues, December 10, 1874.

1866 McArthur, J. A., forfeited membership by non-payment of dues, December 8, 1881.

1861 McCall, C. A., resigned.

1880 McClellan, G., Surgeon to the Philadelphia Hospital, 1352 Spruce Street.

1868 McClure, W. W., resigned September 10, 1878.

1884 McIlvaine, E. S., resigned December 11, 1884.

1864 Mears, J. Ewing, Surgeon to St. Mary's Hospital, 1429 Walnut Street.

1873 Meigs, Arthur V., Physician to the Pennsylvania Hospital, 1322 Walnut Street.

1857 Meigs, John Forsythe, died December 16, 1882. (O. M.)

1874 Miller, C. K. I., died 1878.

1882 Miller, D. J. M., 2310 Spruce Street.

1878 Mills, C. K., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic and School for Graduates, Neurologist to the Philadelphia Hospital, 1909 Chestnut Street.

1885 Mitchell, James, 808 North Sixteenth Street.

1884 Mitchell, J. K., Physician to St. Mary's Hospital, Assistant Physician to the University Hospital, 314 South Sixteenth Street.

1857 Mitchell, S. Weir, Physician to the Hospital for Nervous Diseases, 1524 Walnut Street. (O. M.)

1876 Montgomery, E. C., resigned January 11, 1883.

1857 Morehouse, George R., 2033 Walnut Street. (O. M.)

1885 Morrison, William H., Holmesburg, Pennsylvania. (N. R.)

1887 Morton, S. W., 318 South Fifteenth Street.

1857 Morton, T. G., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)

1885 Morton, T. S. K., 1421 Chestnut Street.

ELECTED

1859 Moss, William, resigned December 10, 1885.

1871 Muhlenberg. Frank, 1911 Chestnut Street.

1869 Müller, August F., 4753 Green Street, Germantown.

1880 Musser, J. H., Physician to the Philadelphia Hospital, Pathologist to the Presbyterian Hospital, Chief of Medical Dispensary, University Hospital, 3705 Powelton Avenue.

1873 Musser, M. B., Obstetrician to the Philadelphia Hospital, 229 South Fortieth Street.

1869 Mustin, J. Burton, died 1871.

1869 Nancrede, C. B., Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic and School for Graduates, Surgeon to the Episcopal Hospital, Surgeon to St. Christopher's Hospital for Children, 2109 Pine Street.

1879 Neff, J. S., 1425 Spruce Street.

1885 Neilson, T. R., 346 South Fifteenth Street.

1880 Newcomet, H. W., died June 1, 1885.

1872 Newsham, Stanley P., resigned December 23, 1881.

1868 Norris, Herbert, resigned January 8, 1880.

1868 Norris, Isaac, resigned November 12, 1874.

1869 Norris, J. C., resigned February 10, 1876.

1861 Norris, William F., Clinical Professor of Ophthalmology in the University of Pennsylvania, 1530 Locust Street.

1875 O'Hara, Michael, Physician to St. Mary's Hospital, 31 South Sixteenth Street.

1886 Oliver, C. A., 1507 Locust Street.

1874 Osgood, Hamilton. (N. R.)

1885 Osler, William, Professor of Clinical Medicine in the University of Pennsylvania, 1502 Walnut Street.

1885 Packard, F. A., Pennsylvania Hospital.

1857 Packard, John H., Surgeon to the Pennsylvania Hospital, 1926 Spruce Street. (O. M.)

1868 Pancoast, William H., Professor of Surgical Anatomy and Clinical Surgery in the Medico-Chirurgical College, 1100 Walnut Street.

1873 Parish, William H., Professor of Anatomy in the Woman's Medical College, 1435 Spruce Street.

1875 Parks, Edward L. (N. R.)

1867 Parry, John S., died 1876.

1870 Paul, Comegys, Belvidere, New Jersey. (N. R.)

1875 Paul, James, resigned January 22, 1880.

1857 Penrose, R. A. F., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)

1865 Pepper, George, died September 14, 1872.

ELECTED

1865 Pepper, William, Professor of Theory and Practice of Medicine in the University of Pennsylvania, 1811 Spruce Street.
1879 Perkins, F. M., 1428 Pine Street.
1885 Piersol, G. A., 1110 Spring Garden Street.
1870 Porter, William G., Surgeon to the Presbyterian Hospital, 1223 Spruce Street.
1886 Powell, William M., 2205 Pine Street.

1885 Rahter, C. A., Harrisburg, Pennsylvania.
1885 Randall, B. A., Ophthalmic Surgeon to the Children's Hospital, 1806 Chestnut Street.
1878 Raub, J. A., forfeited membership by non-payment of dues.
1885 Reed, Boardman, Atlantic City, New Jersey.
1884 Reed, C. H., resigned December 22, 1887.
1874 Reed, H. B., resigned December 9, 1880.
1859 Reed, Thomas B., Surgeon to the Presbyterian Hospital, 1427 Walnut Street.
1871 Rex, George A., 2118 Pine Street.
1869 Rex, O. P., forfeited membership by non-payment of dues, December 10, 1874.
1877 Reynolds, A. S., forfeited membership by non-payment of dues, February 9, 1882.
1864 Rhoads, Edward, died January 16, 1871.
1878 Rich, T. C., resigned January 13, 1881.
1869 Richardson, Elliott, resigned December 8, 1881.
1863 Richardson, Joseph G., died November 13, 1886.
1857 Richardson, T. G., New Orleans, Louisiana. (O. M.) (N. R.)
1884 Ridge, J. M., Camden, N. J.
1886 Riera, J. H., 1630 Vine Street.
1876 Risley, S. D., Lecturer on Ophthalmoscopy in the University of Pennsylvania, 1722 Walnut Street.
1868 Ritz, Charles M. (N. R.)
1886 Riveley, M. P., 121 South Eighteenth Street.
1880 Roberts, A. S., Instructor in Orthopædics in the University of Pennsylvania, 131 South Fifteenth Street.
1876 Roberts, J. B., Professor of General and Operative Surgery in the Philadelphia Polyclinic and School for Graduates, Surgeon to St. Mary's Hospital, 1627 Walnut Street.
1876 Roland, O. (N. R.)
1874 Ronaldson, W. D. (N. R.)
1877 Rudolph, B. J., forfeited membership by non-payment of dues, February 22, 1882.

1867 Santee, Eugene J., 605 North Eleventh Street.
1863 Savery, William, resigned February 11, 1869.

ELECTED

1869 Schell, H. S., resigned December 8, 1881.
1874 Seiler, Carl, 1346 Spruce Street.
1870 Seyfert, T. H., resigned December 27, 1877.
1870 Shaffner, Charles, 251 South Thirteenth Street.
1876 Shakespeare, E. O., Pathologist to the Philadelphia Hospital, Ophthalmic Surgeon to the Philadelphia Hospital, 1336 Spruce Street.
1869 Shapleigh, Elisha B., 658 North Eighth Street.
1868 Sherwood, T. H., forfeited membership by non-payment of dues, December 10, 1874.
1885 Shober, J. B., 1311 Spruce Street.
1878 Shoemaker, J. V., 1519 Walnut Street.
1871 Simes, J. H. C., Professor of Genito-urinary Diseases in the Philadelphia Polyclinic and School for Graduates, Surgeon to the Episcopal Hospital, and to St. Christopher's Hospital, 2033 Chestnut Street.
1883 Simon, J. W. (N. R.)
1868 Sinkler, Wharton, Physician to the Hospital for Nervous Diseases, 1534 Pine Street.
1881 Skillern, P. G., 429 South Broad Street.
1881 Skillern, S. R., 3509 Baring Street.
1858 Smith, A. H., died December, 1885.
1863 Smith, E. A. (N. R.)
1859 Smith, F. G., resigned November 13, 1873.
1876 Smith, R. M., resigned December 9, 1880.
1875 Smith, Stanley. (N. R.)
1876 Smythe, F. G., died 1879.
1871 Sparks, G. W., forfeited membership by non-payment of dues, December 10, 1874.
1866 Spooner, E. A., forfeited membership by non-payment of dues, December 10, 1874.
1871 Starr, Louis, resigned October 14, 1880.
1886 Stewart, David D., 2628 North Fifth Street.
1883 Stiles, G. A., Conshohocken, Pennsylvania.
1857 Stillé, Alfred, Professor Emeritus of the Theory and Practice of Medicine in the University of Pennsylvania, 3900 Spruce Street. (O. M.)
1870 Strawbridge, George, resigned June 10, 1875.
1884 Stritmatter, I. P., 1232 North Fifth Street.
1869 Stryker, Samuel S., Obstetrician to the Philadelphia Hospital, 3821 Walnut Street.

1886 Taylor, L. H., Wilkesbarre, Pennsylvania. (N. R.)
1879 Taylor, J. M., 331 South Sixteenth Street.
1866 Thomas, C. H., resigned February 11, 1886.
1868 Thomson, William, Surgeon to Wills Ophthalmic Hospital, 1426 Walnut Street.

ELECTED

1870 Tinkham, J. H., United States Navy. (N. R.)
1866 Townsend, George D. (N. R.)
1868 Townsend, R. M., died.
1886 Tull, M. G., resigned December 9, 1886.
1864 Turner, A. P. (N. R.)
1863 Tutt, Charles Pendleton, died May 11, 1866.
1863 Tyson, James, Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, 1506 Spruce Street.

1885 Ulrich, W. B., Chester, Pennsylvania., forfeited membership by non-payment of dues.

1871 Valdivieso, R. (N. R.)
1870 Van Harlingen, Arthur, resigned December 8, 1881.
1887 Vansant, Eugene L., 113 South Sixteenth Street.
1879 Van Valzah, W. W., resigned.

1887 Walker, J. B., Obstetrician to the Philadelphia Hospital, 1617 Green Street.
1874 Warder, W. H., Obstetrician to the Philadelphia Hospital, 1212 North Broad Street.
1867 Watson, E. W., forfeited membership by non-payment of dues.
1866 Weightman, J. F., resigned April 27, 1871.
1876 Weir, A. H., resigned April 14, 1881.
1875 West, Franklin, died.
1882 Wetherill, H. M., resigned October, 1885.
1885 Wetherill, R. B., Lafayette, Indiana. (N. R.)
1885 Wharton, H. R., Surgeon to the Children's Hospital, 1405 Locust Street.
1875 Whelen, Alfred, resigned December 23, 1880.
1873 White, J. William, Demonstrator of Surgery in the University of Pennsylvania, Surgeon to the Philadelphia Hospital, 1806 Locust Street.
1868 Willard, De Forest, Lecturer on Orthopædic Surgery in the University of Pennsylvania, Surgeon to the Presbyterian Hospital, 1818 Chestnut Street.
1866 Williams, Horace, resigned December 11, 1879.
1875 Williamson, Jesse, resigned October 13, 1881.
1882 Wilson, C. M., 121 South Seventeenth Street.
1867 Wilson, Elwood, resigned.
1880 Wilson, H. A., resigned December 30, 1882.
1869 Wilson, James C., Physician to the Philadelphia Hospital, Physician to the Jefferson Medical College Hospital, 1437 Walnut Street.
1867 Wilson, James F., 1010 Race Street.
1869 Wilson, J. H. (N. R.)

ELECTED

1873 Winslow, W. H., resigned December 28, 1876.
1864 Wistar, Thomas, resigned February 13, 1867.
1875 Wolford, W. S., forfeited membership by non-payment of dues.
1863 Wood, H. C., Jr., forfeited membership by non-payment of dues, March 23, 1876.
1883 Woodbury, F., 218 South Sixteenth Street.
1865 Woods, D. Flavel, Physician to the Presbyterian Hospital, 1501 Spruce Street.
1855 Woodward, J. J., U. S. A., died 1884. (O. M.)
1872 Worthington, D. J., forfeited membership by non-payment of dues, February 9, 1882.

1886 Young, J. K., Surgical Registrar to the University Hospital, 220 South Sixteenth Street.

1876 Ziegler, G. W. (N. R.)

CORRESPONDING MEMBERS.

1866 Bumstead, Freeman J. Died 1879.

1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York.

1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York.

1885 Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.

1860 Ellis, Calvin. Died 1883.

1858 Flint, Austin, Sr. Died 1886.

1858 Hammond, William A., member of the National Academy of Sciences, New York.

1860 Isaacs, Charles E., Brooklyn. Died June 16, 1860.

1878 Jackson, J. B. S., Boston. Died 1879.

1886 Pye-Smith, P. H., Physician and Lecturer on Medicine at Guy's Hospital, London, England.

1860 Reeves, James E. Wheeling, West Virginia.

1861 Rokitansky, Carl. Died 1878.

1860 Watson, John, New York. Died June 3, 1869.

1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

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CONSTITUTION.

ARTICLE I.

The Society shall be called "THE PATHOLOGICAL SOCIETY OF PHILADELPHIA."

ARTICLE II.

Its object shall be the cultivation and promotion of the study of Pathology, by the *exhibition* and *description* of specimens, drawings, and other representations of morbid parts.

ARTICLE III.

The Society shall consist of *Resident*, *Non-Resident*, and *Corresponding Members*.

ARTICLE IV.

The officers of the Society shall consist of a *President, four Vice-Presidents*, a *Secretary*, a *Recorder*, a *Treasurer*, a *Curator*, a *Committee on Publication*, a *Committee on Morbid Growths*, and a *Business Committee*.

ARTICLE V.

The Society shall be governed by the Code of Ethics adopted by the American Medical Association.

ARTICLE VI.

Amendments and alterations of the *Constitution* and *By-Laws* (heretofore made or to be made) shall require a *two-thirds vote* of a meeting at which there are at least *thirteen members* present; and written notice of the same, signed by three members, must be given at the previous meeting.

BY-LAWS.

SECTION I.

ON THE MEETINGS OF THE SOCIETY.

1. The stated meetings of the Society shall be held punctually at 8 P.M. on the second and fourth Thursdays of each month, except July and August; provided said Thursdays do not fall on legal holidays or Christmas eve.

2. The fourth Thursdays of October and April shall be *Conversational Meetings.*

3. *Special Meetings* may be called by the President when requested by three members of the Society.

SECTION II.

QUORUM.

For the transaction of ordinary business, *six* members shall constitute a quorum; but for the *Alteration of the Constitution or By-Laws*, for the *Election of Officers or Members*, or for the *Expulsion of a Member*, *thirteen* shall be the number required.

SECTION III.

DUTIES OF THE PRESIDENT.

1. The President shall preside over, and maintain order at the meetings of the Society, enforce its laws, and check all irregularities.

2. He shall, when requested by three members of the Society, call a special meeting.

3. He shall be required to sign all orders on the Treasurer, duly passed by the Society.

4. He shall appoint an Auditor at the first stated meeting in October, to examine the report of the Treasurer.

5. He shall, at the annual meeting for elections in October of each year, make an address to the Society, in which he shall describe (sum up) the work of the Society for the past year, and make such suggestions in regard to its labors and the mode of conducting them as may seem to him desirable. This address shall be incorporated in the next volume of the Society's Transactions, and an abstract be published in the journals.

SECTION IV.

DUTIES OF THE VICE-PRESIDENTS.

The Vice-Presidents shall, in the order of their seniority, perform the duties of the President in his absence; and in the absence of these officers, a chairman shall be appointed *viva voce*.

SECTION V.

DUTIES OF THE SECRETARY.

1. The Secretary shall have charge of all the correspondence, and shall take and keep accurate minutes of the proceedings of the Society.

2. He shall notify members of their election, and of the time and place of each meeting, and of any special business to be brought before the Society at that meeting; and shall append to such notice a list of the specimens to be exhibited, as furnished him by the *Business Committee*.

3. He shall prepare and distribute with the notice of the annual meeting at which the election of officers is to take place, slips containing the names of the acting officers. The notices shall be printed on postal cards.

SECTION VI.

DUTIES OF THE TREASURER.

1. The Treasurer shall receive moneys due the Society, and pay them only upon the order of the presiding officer of the meeting at which the appropriation was passed.

2. He shall make an annual report of the funds of the Society at the first stated meeting in October.

3. He shall be authorized to employ a collector, when necessary, to collect dues from members in arrears for more than one year.

SECTION VII.

DUTIES OF THE RECORDER.

1. The Recorder shall make and preserve proper records of the verbal communications and debates, and, in conjunction with the Committee on Publication, shall prepare and arrange for printing these and the written communications furnished by members of the Society. Reports of verbal statements shall always be submitted by the Recorder to the member concerned, before publication.

2. The Recorder shall be, *ex-officio*, a member of the Committee on Publication, and shall receive a salary, to be fixed at the first meeting in October of every year.

SECTION VIII.

DUTIES OF THE COMMITTEE ON MORBID GROWTHS.

1. The Committee on Morbid Growths shall consist of seven members.

2. It shall be their duty to examine microscopically, and report upon all specimens referred to them, and they shall present with their report the microscopic specimen upon which said report is based; and if the preparation is of sufficient interest, it shall be properly labelled, bearing the number of the report, and shall be taken care of by the *Curator* as the property of the Society.

SECTION IX.

DUTIES OF THE BUSINESS COMMITTEE.

1. The Committee shall consist of five members.

2. It shall be their duty to obtain papers on pathological subjects, and to secure and arrange the pathological specimens in the order in which they shall be presented to the Society. All members, as heretofore, possessing the right to present such specimens as they desire, in addition to those provided by the Committee.

3. The list so provided by the Committee shall be forwarded to the Secretary before 12 o'clock on the Monday preceding the meeting, so that it may be furnished to each member on his notice.

4. The Committee shall provide the order of business for the Conversational Meetings.

5. The Secretary and Curator shall be members of this Committee *ex-officio*.

SECTION X.

DUTIES OF THE COMMITTEE ON PUBLICATION.

1. The Committee on Publication shall consist of four members, together with the Recorder.

2. It shall be their duty to examine all papers presented for publication in the Proceedings of the Society, and to decide upon their respective merits; and no paper may appear in the Proceedings which has not been approved by at least two of the Committee.

3. The Committee shall have power to fill its own vacancies.

SECTION XI.

DUTIES OF THE CURATOR.

1. It shall be the duty of the Curator to take charge of such specimens as may be temporarily entrusted to the care of the Society.

2. He shall be authorized to employ a servant, who shall call for specimens at members' houses, take them to the hall of the Society on the day of the

meeting, and return them as soon as possible after the meeting, under such rules and regulations as may be given him by the Curator.

3. He shall also exhibit or cause to be exhibited, such specimens as may be sent to the Society, and shall have charge of the property of the Society.

SECTION XII.

ON THE PRESENTATION OF SPECIMENS AND PAPERS.

1. It shall be the duty of every member exhibiting a specimen to the Society, to furnish previously to the Recorder a full written description of the same, and also to present an abstract (if the paper is of any considerable length) for immediate publication; and any member who neglects to furnish such description, shall not be permitted to exhibit the specimen, unless by the consent of the Society. Members having specimens to present shall notify the Chairman of the Business Committee at least one week previous to the meeting, and this is to be placed on the announcement.

2. Microscopic examinations shall be made, if possible, before specimens are presented, in order that the discussions may be restricted to their proper sphere.

3. The presiding officer may, after an exhibition of any specimen, or the reading of any communication, call upon the individual members of the Society, in succession, for their opinions, or for facts which would throw light on the topic under discussion.

4. The presiding officer may refer any point of doubt, or very special interest, which may arise from any exhibition or discussion before the Society, to a committee to report at the next or an early meeting.

5. No paper read before the Society shall be published otherwise than in the Proceedings, without the consent of the Society.

6. The usual paper read at the Conversational Meeting shall be deferred until the last meeting in October.

7. When a specimen has been reported upon by the Committee on Morbid Growths, as of different nature from that which the title of the specimen as presented indicated, the Recorder is authorized to change the title in accordance with the report of the committee.

SECTION XIII.

ON THE ELECTION OF OFFICERS.

1. All the officers shall be elected *annually*, and may be subject to a re-election.

2. The elections shall take place at the first stated meeting of the Society in October, and the officers shall be elected by a majority of the members present.

3. At the first stated meeting in September, nominations for officers for the ensuing year shall be made.

SECTION XIV.

ON THE ELECTION OF MEMBERS.

1. An election for members may be held at any regular meeting of the Society.

2. Any respectable graduate in medicine, of a school recognized by the American Medical Association, shall be eligible for membership, and need not be a resident of Philadelphia; in this latter case he shall be called a non-resident active member.

3. Candidates (their wish to become members having been previously ascertained) must be proposed in writing by at least three resident members, but such propositions shall not be acted upon by the Society until they shall have been read by the Secretary at two previous stated meetings, nor shall the name of any gentleman so proposed be placed upon permanent record unless he be duly elected.

4. No candidate shall be declared elected unless he receive a *five-sixths* vote. Nor shall any candidate who shall fail of election be eligible for renomination within one year thereafter.

5. All elections to membership shall be declared null if they be not properly acknowledged within a given time after notice has been given to that effect by the Secretary, and this time shall be limited to two months for resident members, three months for corresponding members residing within the limits of the United States, and six months for corresponding members residing in foreign countries.

SECTION XV.

ON THE RESIGNATION OF A MEMBER.

A member may resign only by signifying his desire to that effect in writing to the Secretary, and such resignation shall not be accepted by the Society until he has liquidated all his dues.

SECTION XVI.

ON THE EXPULSION OF A MEMBER.

1. Any member violating the Code of Ethics established by the American Medical Association for the conduct of the members of the medical profession, shall be expelled.

2. The charge or charges of such breaches of professional conduct must be preferred in writing by not less than *three* members of the Society.

3. Due notice of such charge or charges must be given to the member accused at least *one month* before his expulsion can take place.

4. No member shall be expelled except by a *two-thirds* vote at a meeting at which there are at least *thirteen* members present.

SECTION XVII.

ON THE SIGNING OF THE CONSTITUTION.

1. All resident members shall be required to sign the Constitution.

2. The signing of the Constitution and By-Laws adopted in 1857, shall be deemed equivalent to a subscription to the Constitution and By-Laws in force for the time being.

SECTION XVIII.

ENTRANCE FEES AND DUES.

1. The entrance fee to the Society shall be *five dollars*, and each member shall be required to contribute annually *four dollars*, payable the first meeting in January.

2. The contribution of newly elected members to the *annual dues* shall be rated according to the dates of their election, so that members elected in March shall be required to contribute for that year three (3) dollars, those elected in June two (2) dollars, and those elected in October one (1) dollar. Members elected in December shall be liable only for the dues of the succeeding years.

3. Anyone in arrears for more than one year shall *forfeit* his membership, provided he shall have been six months previously notified by the Treasurer of the said indebtedness to the Society.

4. *Corresponding* and *non-resident members* shall be exempt from all fees or dues to the Society.

5. Resident members who leave the city permanently, or those who are absent in the military or naval service of the United States, become non-resident members, and are, as such, exempt from the payment of dues to the Society.

6. Corresponding and non-resident members, on removing to the city, may enjoy the privileges of resident members, by paying regularly the annual subscription.

7. The entrance fee shall be payable upon introduction to the Society, and no member shall be competent to take part in the proceedings of the Society until after payment of such fee.

SECTION XIX.

ON THE INTRODUCTION OF VISITORS.

1. At the ordinary meetings of the Society every member shall be privileged to introduce visitors.

2. All visitors shall retire on the Society's going into private session.

SECTION XX.

ON THE ORDER OF BUSINESS.

The following shall be the order of business for the stated meetings, *except for Conversational Meetings and the first stated meeting in October:*

1. The reading of the minutes of the *general business* of the previous meeting for approval or correction.
2. The introduction of newly elected members.
3. Reports of committees on the general business of the Society.
4. The exhibition of pathological specimens accompanied by written or verbal communications.
5. Remarks on specimens exhibited to the Society.
6. Written or verbal communications on any subject relating to Pathology, and upon these, remarks may be made by members.
7. The Society may then go into private session by order of the presiding officer.
8. Reading the minutes of the *private business* of the previous meeting for approval or correction.
9. Propositions for membership.
10. Election for members.
11. Miscellaneous business.
12. Private business of importance may be called up at any time during a meeting by special resolution.

The following shall be the order of business for the first stated meeting for elections in October:

1. Reading of the minutes of the *general business* of the previous meeting for approval or correction.
2. The Society may then go into private session by order of the presiding officer.
3. Reading of the minutes of the *private business* of the previous meeting for approval or correction.
4. Address of the President.
5. Election for officers.
6. Election for members.
7. Appointment of Recorder's salary.
8. Report of Treasurer and appointment of Auditor.
9. Miscellaneous business.

The order of business for the *Conversational Meetings* shall be provided by the *Business Committee.*

REPORT.

I. THE OSSEOUS SYSTEM.

1. *Abscess and necrosis of tibia.*

Exhibited by Dr. H. Beates, Jr.

The specimen presented this evening is from a strumous gentleman who, three years ago, suffered from an obstinate attack of sciatica, accompanied by a marked degree of muscular atrophy of the affected limb. From the date of this neuralgic affection he was a trifle lame. Two years later a swelling developed at the femoral side of the gluteo-femoral crease, which opened and discharged small fragments of necrosed bone. The sinus was finally healed, and fair health enjoyed for several months. Seventeen weeks ago ostitis of the tibial head developed, followed, in a short time, by suppurative synovitis. The pus perforated the ligamentum posticum, and burrowed beneath the gastrocnemius, elevating it and the posterior tibial muscular structure from the bone. Free incision evacuated this, but the destructive process continued, resulting in the formation of sinuses and complete disintegration of the joint. Hectic was pronounced, and to save life amputation was necessary. With the assistance of Drs. Davis, Dock, Faught, and Mr. Morris, a student in the University, I removed the limb at the middle of the thigh yesterday.

Longitudinal section through the femur, joint, and tibia, disclosed the extensive destruction effected in so comparatively short a time. The articulating surface of the condyles was denuded of its cartilage, and the surface of the bone roughened. The patella was bound firmly to the trochlear portion of the condyles. The crucial ligaments, semicircular fibro-cartilages, coronary ligament—in a word, the articular elements—were destroyed. The head of the tibia was completely necrosed and broken down. The medullary canal of the tibia throughout its entire extent was infiltrated with pus. The surface of the bones was also the seat of the morbid process. The pus from the joint had burrowed upward beneath

the internal and external vasti muscles, and deprived the femur of its periosteum for a considerable distance. The anterior face of the tibia was similarly denuded, while the crest at the middle was the seat of ulceration. An interesting feature is this, on the day the disease occurred the patient first noticed a chancre. Six weeks later the secondary phenomena developed and were promptly met with anti-syphilitic remedies. The question naturally suggests, Was the strumous disease influenced by the syphilis? A microscopical study of the tissues might discover the peculiar arrangements of the cells of the periarterial connective tissue so characteristic of syphilitic arteritis, and thereby furnish positive evidence as to whether or not the specific disease was an influencing element in the cause of the strumous affection. *September* 24, 1885.

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2. *Specimen from a case of gelatiniform arthritis of the knee-joint.*

Exhibited by Dr. H. R. Wharton.

C. M., æt. 5 years, a patient in the Children's Hospital, had suffered from arthritis of the left knee for more than two years. The knee-joint was excised by Dr. John Ashhurst, November 11th. The specimen shows, in addition to ordinary signs of gelatiniform arthritis, a circumscribed patch of caries in the external condyle of the femur, resulting from an osteitis of some standing. *December* 10, 1885.

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3. *Malformation of the hand.*

Exhibited by Dr. H. A. Hare.

Wm. M., colored, æt. 45 years, has had nine children, only three of whom are at present living.

The man has a supernumerary little finger on each hand which is possessed of a nail and is fairly formed. He has no motion in the extra digits, which are apparently not connected with any bony joint surface. The interest in this man's case is increased by the fact that of the nine children which were born to him only three were deformed in the same manner as the father. Nor were the births of the deformed children consecutive. Thus, his eldest child, a boy, possessed six fingers on each hand; the second and third children were normal; the fourth child, a girl, had six fingers like the father, and so did the fifth child, also a girl. The remaining children were normal.

Owing to the fact that the man was at one time a slave, he knows nothing of his parents, and it is therefore impossible to discover whether his extra digits were inherited directly from his parents or not.

Dr. Wharton Sinkler had had a patient under observation some years before, who had a perfectly formed and useful supernumerary third finger, which was, he thought, connected with a supernumerary metacarpal bone. He was one of four sons, three of whom had supernumerary fingers or toes, as also had his father and some of his paternal uncles. There were no deformed females in the family.

Dr. James Tyson recalled two instances of this anomaly: the first in a colored boy, whose father presented the same condition; the second in an idiot with no history of heredity.

Dr. de Schweinitz mentioned a case in which this anomaly had passed through two generations. He also referred to a fœtus, whose cystic degenerated kidneys he had presented to the Society, in which were present on each side six fingers and toes, in which dissection showed the supernumerary digits to be supplied with supernumerary metacarpal or metatarsal bones.

The President had seen several cases of this anomaly in which the supernumerary digit was a mere dermal appendage, situated at the outer side of the hand near the base of the little finger. Two of these instances had occurred in the same family.

Dr. B. A. Randall asked if supernumerary metacarpal or metatarsal bones are often to be found in these cases, a point rather neglected in the previous discussion.

Dr. Wharton had seen a case in which there was a well-marked supernumerary metacarpal bone in connection with a perfectly developed supernumerary finger between the middle and ring fingers.

Dr. H. F. Formad had seen a case in which there was no corresponding metacarpal bone, while in the child's grandfather there was a well-developed supernumerary metacarpal bone. The intervening generation was normal.

Dr. W. E. Hughes said that these, like all other anomalies, are very frequently hereditary, and, again, like other anomalies, are much more common in the colored race. There are all varieties of them, from mere dermal appendages, such as are present in the case to-night, to perfect fingers attached to perfect supernumerary metacarpal bones. Between these are cases in which a perfect supernumerary finger is attached to a normal metacarpal bone by a joint separate from the joint of the normal finger. Very rarely there are supernumerary carpal or tarsal bones.

Dr. Hare said that metacarpal or metatarsal bones are rare; that dermal appendages are quite common; and that bifid members occupy an intermediate position. *December* 10, 1885.

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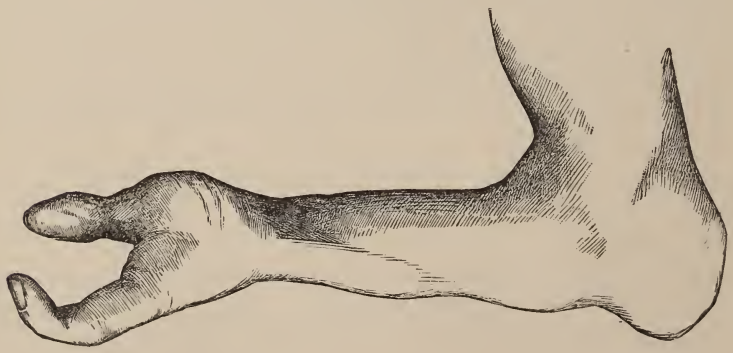
4. *A case of deformity of the forearm and hands, with an unusual history of hereditary congenital deficiency.*

Exhibited by Dr. A. S. Roberts.

Aaron MacI., æt. 73 years, six feet four inches in height, a pedler by occupation, and a native of New Hampshire, was admitted to the Philadelphia Hospital on March 7, 1885. His forearm and hands exhibited the following congenital deformity:

On the right side (see Fig. 1) the humerus is normal, except that its inferior extremity is rounded so that the condyloid notch is scarcely perceptible. In the forearm the ulna is absent, the radius forming with

Fig. 1.



the outer condyle of the humerus an articulation which admits of limited motion in all directions, but which does not admit of either complete extension or flexion. The carpal bones present are those which articulate with the radius, and the first two metacarpal bones. The pisiform, cuneiform, and unciform bones are absent. Of the metacarpal bones, only the first and second are present. The thumb and index finger are present and normal, except that the first phalangeal articulation of the latter is ankylosed, and the finger is as a whole slightly curved toward the thumb. The third, fourth, and fifth digits are wanting.

On the left side (see Fig. 2) no deformity is noticed until we reach the metacarpal bones, except that the olecranon process of the ulna is markedly curved toward the inner condyle of the humerus. Of the

Fig. 2.

metacarpal bones, the third is absent. The first and second each have a thumb, the two thumbs being united by connective tissue and skin, the thumb nails being contiguous, and each complete in itself. The metacarpo-phalangeal joints are enlarged. The index finger is somewhat curved on itself. The middle and ring fingers are absent. This leaves a fissure between the index and little fingers which extends to the

Fig. 3.

wrist, as shown in Fig. 3. The patient has considerable power in his hands, with full use of the parts present.

The following history of similar deformities in his family is interesting, and there is no reason for doubting the veracity of the patient's statements. His grandmother on his mother's side had one index finger stiff. Two uncles on his mother's side had each a stiff little finger. One sister had a hand deformed like the patient's left hand. She had a perfect child. Another sister's child had a hand deformed like his

right hand. The patient has been married twice, and is the father of seven children. The two by his first wife were perfect. Of the five children by his second wife, two were perfect and three were deformed with malformations similar in character to his own.

Although many cases somewhat similar to, or resembling the one above described have been reported, principally by Annandale, the unusual heredity observed in the present instance was deemed worthy of more than passing notice. Here heredity seems to have assumed unusual prominence, several generations having reproduced at one time or another malformations, which, although not absolutely similar, have been markedly so in the type and locality of the deformity. In studying the etiology of congenital malformations we find the subject surrounded by much that is mysterious and confusing, owing to the varying importance attached by authors to the influence of physical and psychical conditions of the patients upon the child in utero. I will not attempt to deal here with the very extensive question of predisposition to or inheritance of disease in general, but only briefly consider malformations, and especially those due to arrested development.

Hereditary similarities have been observed alike in the most extensive and most minute forms. These may vary from the shape of a particular part of the body, or of a special organ, such as the nose or ear, to the small pigment stain known as a "mother's mark." Accompanying these physical marks, similar mental tendencies and physical carriage can be observed for successive generations, giving to races and persons their marked individuality. In the same way that organs perfectly normal, but characteristic, are reproduced, physical defects and abnormalities are transmitted from parent to child. Most congenital malformations, especially those due to arrested development, have been referred to pathological changes affecting the child in utero. It being granted that the fœtus has its own circulation and nutrition, and that, consequently, disease of a part may take place resulting in effusions, exudations, atrophies, and hypertrophies, with consequent marked nutritive changes, this will not account for all the malformations met with. For whilst these lesions undoubtedly cause certain deformities, it has been found experimentally, according to Wagner, that malformations may be produced by wounding the ovum, and thus preventing the development of the part implicated.

Thus the action of mechanical agencies, such as blows and falls at an early period of gestation, have a claim as causative agents in the production of anomalous development. The question of maternal im-

pressions, through the influence of fright, shock, etc., is also to be considered, and their possibility as factors cannot be entirely denied on *a priori* grounds, the literature of the subject abounding in many instances where the relation of cause and effect is seemingly very clear. The result of the most recent investigations, however, would tend to show that the effects apparently brought about by these, are the results of fœtal disease or spontaneous amputations. Simpson and Montgomery have described cases where portions of the digits and extremities have been so amputated, and Simpson has called attention to certain rudimentary digits sprouting out from the end of the stumps. These effects have also been demonstrated by preparations, where it is shown that by the encircling of the extremities by turns of the umbilical cord, or by bands of false membranes, spontaneous intrauterine amputations have resulted. Lastly, slight lesions in undeveloped fœtal organs can cause great disturbances by preventing proper nutrition of the parts and thus the progress, size, and quality of the organ being interfered with, the resulting adult development is either defective or entirely wanting.

These, in brief, are the causes of congenital malformations, and while many of the questions in regard to this class of cases are still in doubt, sufficient etiological explanation can be deduced in the reasons already stated for most of the cases met with, without resorting to apparent coincidences or fanciful theories. *December* 10, 1886.

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5. *Caries of the spine.*

Exhibited by Dr. M. H. Fussell.

The subject from whom the specimens were removed, presented herself as a patient at the University Dispensary in June last, with the following history:

Ida G., æt. 13 years, native of Philadelphia. Father died of phthisis. Has an aunt suffering from Bright's disease. Four years ago began to fail in health; was never confined to bed, however; never noticed any undue prominence along the spine. About three years ago a swelling appeared in the right groin, which finally opened and ever since has been discharging pus. About one year ago a large ulcer, of the size of the palm of the hand, formed over the upper lumbar vertebræ; this has never healed. About six months ago the feet, hands, and face began to swell.

Condition at first visit.—All the tissues of the surface œdematous; face so much swollen that the eyes were almost closed; hands, feet, and limbs pitted deeply on pressure; abdomen prominent and filled with fluid; small amount of fluid in each pleural cavity; in right groin was the pouting orifice of a fistula, from which flaky pus was discharged; over the vertebræ was the ulcer noted above; heart sounds normal; passed but a small amount of urine, which was loaded with albumen and contained numerous waxy and hyaline casts. Patient was seen at home and abdomen tapped, from which one gallon of straw-colored liquid was withdrawn; on standing, this liquid did not solidify and precipitated but little sediment. After the tapping the point of puncture did not close, but liquid discharged from it during the remainder of life. For about one month after being tapped the patient improved somewhat. Then, after almost all the anasarca had disappeared, she ceased to secrete urine and died of uræmia.

Autopsy.—Body much emaciated; no œdema except in lower limbs; abdomen prominent; in the groin the fistulous opening above noted. In the linea alba, just below the umbilicus, was the opening made by the trocar at the time of tapping. A probe passed into the fistulous opening passed upward and backward for five inches. A probe passed into the puncture in the linea alba moved freely in the abdominal cavity. On opening the abdomen a quart of straw-colored liquid escaped. In each pleural cavity was a pint of fluid. Lungs were crepitant throughout. At the apex of the right lung were a few star-shaped small cicatrices. Bronchial glands were enlarged and calcified. There was no excess of fluid in the pericardium. Heart muscle was pale, rather flabby; valves normal. The parietal peritoneum was covered with soft yellowish miliary bodies, while the visceral peritoneum was free from these bodies. Intestines were normal, with the exception of the cæcum, which was abnormally adherent. Spleen of about normal size, hard, and on section of dark-red color, showing plainly the sago grains; on spreading iodine over the surface, these bodies took on the characteristic color of amyloid degeneration. Liver weighed two pounds, soft, rather lighter colored than normal; surface for the most part smooth, except in the middle of the surface of the right lobe, where there were a few miliary bodies. Iodine did not show any amyloid change. Both kidneys were glued to the abdominal wall by inflammation of the surrounding tissues. This was most marked on the right side, where a great mass of inflammatory tissue surrounded the kidney. Both kidneys were enlarged and of a uniformly white color; the capsules stripped off easily; the primary lobules showed with unusual distinctness. On section the cortex was

much thickened, of a uniform white color, the pyramids of a pale reddish color. Pelves of both kidneys were filled with creamy pus, and the lining membrane was the seat of inflammation, being of a bluish-red hue. Both ureters were patulous Iodine showed amyloid degeneration. The iliac fascia was much thickened, and a probe introduced into the opening in the groin could be felt through the much thickened tissue passing up toward the spine. Cutting down upon this probe it was found lying in a track about a quarter of an inch in diameter near the spine, gradually enlarging toward the groin, where it was about half an inch in diameter. The walls of this channel were about a quarter of an inch thick. Tracing this probe up toward the spine it was found to come in contact with carious bone, which was apparently confined to the posterior and lateral parts of the last dorsal and first lumbar vertebræ. There was no irregularity along the line of the bodies of the vertebræ. Microscopical examination showed the miliary bodies on the peritoneum to be of a tuberculous character. The liver had undergone a slight degree of fatty degeneration, but no amyloid change could be seen. Spleen and kidneys showed amyloid degeneration. The latter showed also considerable increase in the connective tissue, and some proliferation of the epithelium of the tubules. *January* 28, 1886.

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6. *Congenital anomaly of both hands.*

Exhibited by Dr. F. A. Packard.

The man presenting the following condition is one of a family of five children. When I first saw him I thought that he had had performed an amputation of both middle fingers with their corresponding metacarpal bones; but on questioning him and on closer examination found the following extensive abnormality:

The right hand consists of a thumb, an index and a little finger, with only two metacarpal bones.

The thumb is about one inch and a half long, consists of two phalanges, and articulates with the same metacarpal bone as the index finger, forming with the metacarpal bone an oblique angle of about 130 degrees. The power of flexion is absent; that of extension slight. Nail present but stunted.

The index finger measures two and a half inches in length and is very thick. It has three phalanges, the distal phalanx being very small as

compared with the others. Last phalanx forms an angle of about 20 degrees with the second phalanx. The nail is stunted. The finger can be flexed as a whole, but the individual phalanges are fixed. The power of extension is slight.

The middle and ring fingers are totally wanting.

The little finger measures three inches in length. There are three phalanges, the distal one being small. The last two phalanges form a right angle with the proximal phalanx. The nail is stunted. The finger can be flexed and extended as a whole, although the individual portions are rigid. The metacarpus is represented by two very large, heavy bones, that are separated from each other by an angle of about 45 degrees. Two metacarpal bones both articulate with the carpus, but not with each other.

The left hand consists of a thumb, index, ring, and little fingers, with four corresponding metacarpal bones.

The thumb is normal in size and proportions, with perfect nail and normal range of motion.

The index finger is large, three and a quarter inches in length, and has three phalanges. The last phalanx is very small and the nail is stunted. The two distal form a right angle with the proximal phalanx. There is moderately good range of flexion and extension.

The middle finger is absent.

The ring finger is three inches long and has three phalanges. The last phalanx is small, with a very fairly developed nail. The last two phalanges form a right angle with the proximal phalanx. Range of motion is good.

The little finger measures one and a half inches in length and has three phalanges. Nail small.

The metacarpus is normal, except for the fact that instead of a metacarpal bone to correspond to the absent middle finger, there is an angle of about 30 degrees between the metacarpal bones of the index and ring fingers. The metacarpal bone of the little finger is rather small, and is close to, but does not articulate with that of the ring finger.

The index and ring fingers with the thumb can be closely approximated, and the index and ring fingers can be so moved as to close in the angle formed by the absence of the middle metacarpal bone.

To abbreviate, there is the following condition:

Each hand, considered as a whole, consists of two portions separated from each other by a considerable angle. One portion of the right hand is composed of the thumb and index finger, with a common metacarpal bone, the other of the little finger with a metacarpal bone. One portion

of the left hand is formed by the thumb and index finger with corresponding metacarpal bones, the other portion is composed of the ring and little fingers with their separate metacarpal bones. The angles between the two portions in each hand run completely down to the wrist.

The man's mother and father are normal as regards their hands. One sister has one hand that resembles his left. His other sister has a left hand similar to his, and on the right hand has the middle and ring fingers united throughout their length. One brother has both hands similar to this man's left hand. The only member of his generation not deformed is his *twin* brother, who has normally developed hands. He has several children, none of whom present any peculiarities of structure, and the same is true of his nephews and nieces.

He knows of no maternal impression that could have borne any causative relation to the deformity, nor does it seem certain that heredity has had anything to do with it, although he says that he thinks he has heard that his father's grandfather had some peculiarity of his hands.

His hands have always been deformed to as great an extent as at present. He is a farmer by occupation, and has never felt any inconvenience or discomfort from his deformity. He can perform all kinds of farm work apparently as well as normally constituted men.

I had intended to obtain a cast of the hands, but unfortunately only saw the man on one occasion, and was not prepared to do so at that time. *March* 25, 1886.

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7. *Specimen from a case of compound depressed fracture of the skull, with a wound of the lateral sinus.*

Exhibited by Dr. H. R. Wharton.

The patient, M. M., æt. 24 years, from whom this specimen was removed, was admitted to the University Hospital on the evening of August 30, 1886, with a compound depressed fracture of the occipital bone on the right side on a line with the occipital protuberance. He had received the injury while at work upon a railroad by being struck by a dump cart. He had also received a severe contusion of the chest, and when I saw him, shortly after his admission to the hospital, he was conscious and exhibited very marked symptoms of shock; he also suffered from dyspnœa, and auscultation over the left chest showed diminished respiratory murmur.

Such being his condition, and as he presented no well-marked symptoms referable to his head injury, and as I suspected a rupture of the left lung, I deemed it inadvisable to interfere with the depressed fracture at that time. His general condition improved by the next day, and he was seen by Prof. Ashhurst, who elevated and removed the depressed fragments of the occipital bone. Upon the removal of the largest fragment, the location of which corresponded very closely to the position of the junction of the longitudinal and lateral sinuses, there was a profuse gush of venous blood; this was instantly controlled by packing the wound with antiseptic gauze and the application of a compress and bandage.

The patient made a good recovery, and was discharged from the hospital at the end of six weeks, the packing having separated and the wound being solidly healed by that time. During the month of February following, he was again admitted to the hospital, suffering from typhoid fever, from which he died a few days after his admission.

Through the courtesy of Prof. Osler, in whose ward the patient was, I have the opportunity of exhibiting to you the specimen which shows the extent of his head injury and the amount of repair that has taken place. You can see that the loss of bone resulting from the removal of the depressed fragments has been filled by a firm fibrous membrane and also upon the inner surface of the specimen you can see a large well-organized thrombus occupying a portion of the lateral and longitudinal sinuses. I also present the fragments of bone removed shortly after his first admission to the hospital. The specimen is of especial interest, for we rarely have the opportunity of seeing the condition of the parts so soon after recovery from an injury of this nature. It also exemplifies the truth of the surgical dictum that wounds of the venous sinuses of the brain should be treated as wounds of large venous trunks in other parts of the body.

The treatment employed in this case, packing the wounded sinus with strips of gauze proved eminently satisfactory, and is at the present time, I think, the procedure generally recommended in this class of injuries.

The course of this case also coincides with the clinical observation that has been made, that a wound of the venous sinuses of the brain is not so immediately dangerous to life when the wound communicates with the air through a wound in the skull and soft parts, for when these conditions do not exist, pressure symptoms from the accumulation of blood generally supervene rapidly. *June* 10, 1886.

8. *Bony mass removed by incision from the knee-joint.*

Exhibited by Dr. H. R. Wharton.

P. C., æt. 36 years, was admitted to Prof. Ashhurst's ward in the University Hospital, October, 1886, complaining of a painful condition of the right knee-joint, which came on suddenly and utterly incapacitated him from following his ordinary avocation, which was that of a miner. He attributed his disability to the presence of a movable body in the right knee-joint which appeared sometimes to the outer, sometimes to the inner side of the joint.

He had experienced the same trouble and noticed the presence of the movable body sixteen years before, but the trouble then lasted only a short time, and up to the present attack, six weeks before his admission to the hospital, he had suffered no inconvenience.

The specimen which I present to-night was removed by Prof. Ashhurst at his clinic, by a small incision over the lower part of the external condyle of the femur, being first transfixed through the skin by a needle. It was found attached by a pedicle, upon dividing which, the mass was removed without difficulty; the wound healed promptly, and the patient made a good recovery.

The name loose cartilages is generally applied to these movable bodies found in the joints, although after removal they are as often found to consist of bony or fatty tissue as of true cartilage.

A point of interest in their clinical history is the fact that there are intervals during which they cause the patient no inconvenience, as was markedly the case in the present instance.

The old writers were inclined to attribute presence of movable bodies in the joints to the organization of a blood-clot resulting from an injury to the joint, but it is now thought that they originate from the villous finger-like processes of the synovial membranes of the joints; they may be attached by a band of narrow pedicle or be entirely detached and free to move in the articular cavity.

Paget has described cases in which the movable body appears to have been a portion of the articular cartilage of the joint separated by a localized quiet necrosis. The favorite location for the presence of these bodies is in the knee-joint, fully eighty-five per cent. of the reported cases having been observed in this joint. *November* 11, 1886.

9. *Conical stump.*

Exhibited by Dr. H. R. Wharton.

The specimen consisted of a portion of a humerus removed from a boy æt. 14 years, by reamputation. The appearance of the parts was most characteristic. Dr. Wharton remarked that amputation through the humerus or tibia in children is likely to be followed by the formation of a conical stump, owing to the fact that the growth of these bones in length is largely from the upper epiphysis. *January* 27, 1887.

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10. *Specimens of chronic knee-joint disease.*

Exhibited by Dr. H. R. Wharton.

The specimens present the characteristic appearances which are so commonly observed in advanced cases of this disease.

At the present time I think the tendency is to attribute all of these cases of strumous or gelatiniform disease of the knee-joint to a tuberculous origin.

The new tissue in these cases does not tend to cicatrization or fibrillation, but tends to remain indefinitely the same unformed embryonic material, and is prone to undergo fatty, purulent, or gelatiniform degeneration. The extreme obstinacy in the reparative process in these cases of knee-joint disease has been attributed to the presence of tubercle in the synovial membrane, cartilages, and bone, and its presence has unquestionably been demonstrated in certain cases.

But it is a sufficiently well-known fact that inflammatory products, if sufficiently persistent, are apt in strumous subjects to become tuberculous.

I should, therefore, hesitate to classify all of these as cases of tuberculous arthritis of the knee-joint, unless the presence of tubercle could be demonstrated.

I have observed clinically that disease of the knee-joint is not so apt to be followed by the development of tuberculous affections of the internal organs as in cases of hip-joint disease or caries of the vertebræ.

Two of the specimens, one from an adult, the other from a boy æt. 12 years, were taken from cases which had advanced well toward a cure by bony ankylosis of the knee-joint with the limb in bad position, and the operation was undertaken in these cases to correct the deformity

by the removal of a wedge-shaped piece of bone which included the patella and a portion of the tibia and femur.

The specimen of knee-joint disease which was removed by amputation of the thigh, shows such extensive disease of the bones and soft parts that the more conservative operation of excision was rejected, and amputation at the lower portion of the thigh was resorted to.

February 10, 1887.

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11. *Specimens from four cases of necrosis of the jaw occurring in children.*

Exhibited by Dr. H. R. Wharton.

These specimens were removed from patients in the Children's Hospital by Prof. Ashhurst and myself. One specimen is from a case in which the disease involved a portion of the upper jaw only, following an attack of measles. In another case the specimen consists of the greater portion of the lower jaw. The disease in this case was so extensive that the whole of the lower jaw was removed with the exception of the ascending ramus and condyle on the right side. The disease followed an attack of measles. The third specimen is from a case in which a portion of the lower jaw only was involved, following an attack of measles. The specimens in the fourth case were removed from a case in which there existed a disseminated necrosis involving both jaws, symmetrical portions of both upper and lower jaws being involved. In this case the affection of the bone is said to have followed an attack of ulcerative stomatitis. These cases represent a variety of necrosis of the jaw occurring in children as sequelæ of measles, scarlet fever, and smallpox, and to which the name exanthematous necrosis has been applied by Salter. Gangrenous stomatitis, which is itself a not infrequent sequelæ of the above-named diseases, I have frequently seen as a cause of necrosis of the jaw in children. It is very probable that many of the cases of necrosis of the jaw which were formerly attributed to the use of mercury were really cases of exanthematous necrosis, and were not due to the administration of that drug.

March 10, 1887.

12. *Specimens from six cases of excision of the elbow-joint.*

Exhibited by Dr. H. R. Wharton.

Dr. H. R. Wharton presented six specimens from cases of excision of the elbow-joint removed from patients in the University and Children's Hospitals. In four of the cases excision was performed for scrofulous or tuberculous arthritis of the elbow-joint, three of the patients being children, one being an adult. The results of the operation were satisfactory, except in one case in which death resulted several months afterward from tubercular meningitis. In one case the specimens were removed from the arm of a man who had sustained a railroad crush of the elbow involving the joint. In this case a partial excision of the joint was performed instead of amputation, as the patient had lost his other arm from gunshot injury a few years before. This patient died a few days after the operation from traumatic spreading gangrene. In another case the specimens were removed by Prof. Ashhurst, in the Children's Hospital, from a child aged six years suffering from syphilitic arthritis of the elbow-joint. In this case the disease had probably had its origin in a gummatous affection of the bursæ in the neighborhood of the joint, which had gradually involved the articulation. The patient from whom this specimen was removed suffered also from syphilitic pannaris, and had also several ulcerated gummata on other portions of the body.

March 24, 1887.

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13. *Sarcoma of the upper jaw.*

Exhibited by Dr. H. R. Wharton.

Dr. Wharton presented a large tumor removed from the upper jaw of a patient æt. 50 years, by Prof. Ashhurst at the University Hospital on April 10th. The patient stated that the growth had begun only three months before he came to the hospital, and had attained its present large dimensions in that space of time. The whole of the left superior maxillary bone with its orbital plate was involved in the growth and was removed. The growth is probably a sarcoma. The patient has done well since the operation. *March* 24, 1887.

14. *Fracture of the skull from gunshot injury.*

Exhibited by Dr. H. F. Formad.

The patient, a negro man, from whom the specimen was obtained, had been struck in the middle of the forehead by a pistol-ball, had fallen immediately, and died after a short period of unconsciousness. On examination it was found that the ball had pierced the skull anteriorly, making a clean-cut, square hole, passed directly through the brain, pierced the skull again posteriorly, and lodged beneath the scalp. Death had resulted from hemorrhage in the track of the ball. The point of entrance of the ball was exactly in the middle line of the frontal bone. The point of interest about the case is the fact that there was a fracture extending in a direct line clear through the median line of the frontal bone and back to the occiput, as though the parts had been riven apart by the ball acting as a wedge. Dr. Formad questioned whether the fracture might not have been the result of the fall rather than of the gunshot injury. *May* 12, 1887.

II. THE DIGESTIVE APPARATUS.

1. *Primary carcinoma of the liver.*

Exhibited by Dr. W. E. Hughes.

. The woman from whom the specimens were obtained was a patient of Dr. Whiteside, to whom I am indebted for the following clinical history: White, æt. 58 years; a daughter had died of cancer of the uterus; remainder of family history unknown. She had been in good health till eighteen months ago, when she began to have occasional attacks of what she thought sick headache. For a day or two she would have sharp headache accompanied by excessive nausea and vomiting, then these symptoms would pass off and give place to a diarrhœa lasting sometimes a week. In addition to these attacks there were irregular paroxysms of sharp pain in the hepatic region, and her flesh and strength failed progressively. With the exception of the sick headache her digestion seemed good. Dr. Whiteside saw her for the first time last June. She was then deeply jaundiced (her first attack), and was in one of her spells of headache. These had been occurring at intervals of a month to six weeks, and had been increasing in frequency and severity. She was much emaciated, and complained of extremely severe sharp pain in the region of the liver. On examination, this organ was found enlarged, and projecting from its surface could be felt, just below the margin of the ribs, a firm, hard, rounded mass rather larger than a hen's egg. The jaundice cleared up in two weeks, her digestion improved, and she was able to take a fair amount of nourishment, principally liquid; but her liver steadily enlarged, and emaciation progressed. There was no return of jaundice. She died November 8th of exhaustion, after a week of uncontrollable vomiting and diarrhœa.

Autopsy, thirty-six hours after death.—Body terribly emaciated. Spots of chloasma over the surface, but no jaundice. The enlarged and irregular liver could be plainly seen through the abdominal parietes. On opening the body the tissues were found almost bloodless. Adipose tissue almost completely wanting. Liver was uniformly enlarged; scattered through its substance were numerous cancerous nodules varying in size from an orange to a scarcely perceptible point; those projecting from the surface were

characteristically cupped. The largest growth, apparently the primary one, was situated in the upper part of the right lobe. On section the nodules showed a pinkish succulent surface with no degeneration, and shaded gradually into the liver substance. In addition to the cancerous nodules were several cysts filled with clear fluid, varying in size from a pea to two inches in diameter, the largest one in the left lobe. The liver substance itself was normal. The ductus communis choledochus was patulous. The retroperitoneal and mediastinal glands were a little enlarged, and evidently the seats of beginning cancerous deposits; no enlargement of the glands in the fissure of the liver. The intestines were pushed well to the left half of the abdominal cavity by the enlarged liver; the large intestine was empty and flaccid; the small intestine moderately distended with fluid. At the ileo-cæcal valve was an intussusception three inches in length, reducible with some difficulty, and after reduction showing the apposed peritoneal surfaces covered with a thin layer of lymph. The intussusception apparently permitted of the passage of no liquid. The stomach was pushed downward, and was very small, measuring at its widest part not more than one inch in diameter; its walls were not unduly thickened, and its mucous membrane apparently healthy. Brain and spinal cord not examined. Other organs not remarkable. Careful search failed to reveal any growth outside the liver, except the small secondary deposits in the glands above mentioned. *November* 12, 1885.

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2. *Cancer of lower third of œsophagus; metastasis to stomach and liver.*

Exhibited by Dr. T. S. K. Morton.

The notes of the case from which these specimens were obtained, are as follows:

Annie C., æt. 61 years, domestic, a native of Pennsylvania. Her father was killed, mother died of phthisis, brothers and sisters are healthy. Sexually she was normal; the mother of four children; no miscarriages; menopause at forty-five. Only previous disease was pneumonia of right side, one year ago. She had always been of a bilious disposition, and the present illness began nine weeks before admission to the Pennsylvania Hospital, with a severe attack of jaundice, nausea, which gradually developed into constant emesis, acute lancinating pain in right hypochondriac region, shooting to the umbilicus, anorexia and constipation; she had passed no feces for the preceding five weeks.

Had noticed "coffee-ground" matters in vomit, several times, but never bright blood. Cough and respiration gave rise to epigastric pain.

Upon admission she was found to be exceedingly emaciated and sallow, complaining of great pain, as above. Tongue pale and heavily coated with brownish-white moist fur. Urine: sp. gr. 1018; no sugar or albumen, but contained traces of bile. Lungs and heart normal, except for what proved to be anæmic murmurs in the latter organ. The abdomen was very flat; the aorta and vertebral column could be felt upon slightest palpation. The pylorus could readily be felt lying across the aorta just above the umbilicus; it was normal in size and shape, but was exquisitely sensitive to pressure.

The intestines were much contracted—some feces could be felt in the descending colon. Vomited matters consisted entirely of food, mucus, and epithelium, no blood at any time. The glands of the lateral cervical regions were enlarged and very hard. In spite of all known remedies for vomiting, and constant rectal alimentation, she steadily sank, and died twelve days after admission.

Post-mortem examination.—Beginning at the lower third of the œsophagus, and extending to the cardiac orifice of the stomach, and also involving the neighboring retroperitoneal glands, and, to a slight degree the head of the pancreas, was found a cancerous mass surrounding and narrowing, but by no means obliterating the œsophageal lumen.

Neither the pancreatic or biliary ducts, nor any bloodvessels were in any way involved.

There was no dilatation of the œsophagus above the cancer. Many small metastatic cancer nodules were found in the liver and stomach, in the former organ principally in the Spigelian lobe. All of the other organs were small though entirely negative, and no additional metastases were found.

The peritoneal glands and even those of the neck were somewhat enlarged and very hard.

Microscopic examination of sections of the above cancer, metastases, and infiltrated glands prove it to be of the hard variety or scirrhus.

January 14, 1886.

3. *Cirrhosis of liver and perihepatitis.*

Exhibited by Dr. T. S. K. Morton.

The case from which this liver was removed was that of a woman, æt. 40 years, a former circus-rider, who had always been accustomed to large amounts of the strongest spirits. She became ill first when thirty-five years of age; having slight ascites and œdema of lower extremities. For this she treated herself with large amounts of gin.

When admitted to the Pennsylvania Hospital, in August last, her symptoms were marked ascites and œdema of legs, abdomen tense, and superficial venous anastomoses very markedly developed, much dyspnœa, some cyanosis, tenderness over splenic and hepatic regions, and diminished liver area. Heart, lungs, and urine were normal. She remained in the institution four months and died finally from an intercurrent attack of adynamic facial erysipelas; the temperature being subnormal throughout, except during the first few hours. During these four months it became absolutely necessary to tap the abdomen eight times, the average amount of liquid withdrawn being six quarts. Albumen and casts in profusion appeared in the urine with the onset of the erysipelas. After death the liver was found to be much cirrhosed, and perihepatitis developed to a marked degree. The organ weighed but thirty-six ounces, and was bound down by cicatricial adhesions in all directions, more especially at its diaphragmatic surface, and about the portal vein, which latter was stenosed as it entered the organ and much dilated below that point. The kidneys presented parenchymatous inflammatory changes, but the necropsy was otherwise negative. *January* 14, 1886.

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4. *Amyloid disease following chronic dysentery.*

Exhibited by Dr. T. S. K. Morton.

Mary B., æt. 20 years, white, single, a native of Ireland, was admitted to the Pennsylvania Hospital, December 18, 1885. The family history was excellent, both parents and a number of brothers and sisters are living and healthy. She, herself, was likewise, in the enjoyment of excellent health, she stated, until two years ago. At that time her menstrual discharge, which had commenced when she was seventeen, abruptly dis-

appeared; and had not recurred at any time since. Shortly after its cessation she began to feel ill, but was able to do light work until last winter when she entirely broke down. Thinking that a sea voyage and change of climate would be beneficial, she engaged passage for America, landing at this port early during the past summer.

For a short time after this she seemed improved, but, acquiring whooping cough, she spent the entire summer in the University Hospital under treatment for it and its consequences. During this time she lost much flesh and strength.

Inquiry of Dr. Rhoads, the resident physician, under whose care she was while in the above institution, developed the following facts: "She had marked whooping-cough with many diffused bronchitic and asthmatic râles, and these physical signs continued for a long time after her cough had mainly subsided, or at least had lost its specific character, so that I feared that phthisis might develop. She had no marked abdominal symptoms so far as I can recollect. She was troubled, mentally, by amenorrhœa, her menstruations having been absent for a year or two, though she affirmed she had menstruated at one time, but her menstruation had ceased two years after its commencement." Since leaving the University Hospital she stated that she had been slowly improving till three weeks ago, when she again began to feel poorly, and that shortly afterward fever and a profuse diarrhœa set in, which have lasted since, and still continue, the stools being frequent, profuse, liquid, and often blood-stained in varying proportions. She had never had much headache or emesis, nor had she ever had epistaxis or other hemorrhages, chills and fever, rheumatism, or any other sickness that she could remember. Since last summer's attack had had a very slight cough, but no expectoration; she never sweated. Eyesight and hearing were normal; memory very defective. The present sickness had been attended with little or no pain; what little there was had been confined to the abdomen. She had never had any œdema, palpitation, or dyspnœa. Everything that she had taken of late had instantly "gone through her." Did not recall any previous attacks of diarrhœa.

Upon admission she was much emaciated, and very weak, sordes were abundant, also herpes labialis. The tongue was dusky-red and dry, while both it and pharynx were aphthous. She was extremely thirsty. Skin was dry, hot, and very harsh, while remnants of sudamina, but no other eruption was present over the abdomen. The mind was clear, but the memory could scarcely be trusted. She had total anorexia and some headache. Heart and lungs normal, although the respiratory sounds upon the right side were replaced to a high level by the liver. Examina-

tion of abdomen showed much dilated superficial venous anastomoses; tenseness of abdominal walls; fulness of the cavity, with slight tympany; liver dulness extended to the umbilicus; gall-bladder could not be felt; spleen enlarged. The liver area was tender upon palpation, and she stated that more or less pain had been present there for a number of weeks, though much worse during the last two. She passed many copious, rather fetid, dark-colored, liquid stools, which were streaked and tinged with arterial blood. Red corpusles were diminished six per cent., while the white corpuscles were very markedly increased. Vaginal and rectal examinations were entirely negative. Stools contained, as shown by the microscope, blood, pus, and some free fat and unaltered food. The urine had a specific gravity of 1.005, contained no sugar but much albumen and granular and epithelial casts. Temperature 100.4° F.; pulse and respiration to correspond.

Progress: Notwithstanding the active employment of a great variety of medicines she continued until the end to have from four to ten of the above-described stools daily. Vomiting also occurred from time to time, as did pain in the hepatic and lower abdominal regions.

Dec. 22. Has failed much in flesh and strength; epistaxis to-day.

26*th*. Covered with sudamina.

29*th*. Stools extremely offensive. For about a week she has gradually been becoming icterode, more especially so to-day; the skin itches much.

Jan. 11. Died of exhaustion. At none of the many examinations of the urine were amyloid casts found. Since admission steady pyrexia had been constantly present; it usually varied from 100° to 103°, having only once or twice touched almost the normal.

Autopsy.—Brain and spinal cord not examined. Thoracic contents normal. Liver, spleen, and kidneys, all much enlarged and infiltrated with amyloid material; the kidneys also showed parenchymatous inflammatory changes. The large bowel throughout was in a state of chronic dysenteric ulceration, worm-eaten in character and typical. A mass of glands in the left axilla, which were noticed when she was admitted and supposed to be œdematous, could not be obtained. For the privilege of presenting this case I am indebted to the kindness of my chief, Dr. James H. Hutchinson.

Dr. James Tyson said this was the first case he had ever met with in which there was this association of amyloid change with dysentery as the etiological factor, although this seems to be the only possible cause in this instance where the possibility of syphilitic disease seems excluded.

Dr. W. Osler thought it was well recognized that chronic dysentery might be followed by extensive amyloid disease. He had met with one

or two instances, in connection with chronic diarrhœa, which post-mortem examination showed dependent on chronic dysentery, with very much the condition of bowel as present in Dr. Morton's specimen.

January 14, 1886.

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5. *Hemorrhagic pancreatitis, with swelling of the semilunar ganglia and Pacinian corpuscles.*

Exhibited by Drs. W. Osler and W. E. Hughes.

The specimens were removed from the body of a man, æt. 58 years, whose lungs were emphysematous to a remarkable degree, and whose body presented slight œdema, accompanying the heart trouble due to emphysema. The Pacinian corpuscles, about forty in number, were found in the connective tissue behind the duodenum and pancreas, showing as bluish-white, translucent, rounded, ovoid, or reniform bodies, with a central white axis. They varied in size from the largest, 6 mm. long by 4 mm. thick, to some not larger than normal. They were all plainly attached to nerve filaments, most of them scattered and single, a few in groups of three or more. The increase in size was due to an œdematous condition of the concentric sheaths. This form of enlargement of the Pacinian corpuscles seems to have almost completely escaped observation, for it is not referred to in any of the text-books on morbid anatomy; but Dr. E. Przewoski, in Virchow's *Archiv*, Bd. lxiii., describes five cases in which these bodies were affected in this way. In two the subjects were œdematous, and it is probable that, if looked for, they would not infrequently be met with. The semilunar ganglia, though to the naked eye large and succulent, presented under the microscope no decided lesion, only an increased number of small round cells, with a cloudiness and indistinctness of the ganglion cells.

The pancreas presented an interesting lesion. Everywhere the connective tissue was filled with extravasated blood. Especially was this the case toward the tail, where there were even actual clots. In the body of the gland the acini themselves were not involved, but appeared as grayish-white areas, surrounded by a dark-red hemorrhagic exudation. The loose tissue in the neighborhood was infiltrated with serum. Klebs gives a good account of hemorrhage into the pancreas, and has collected several cases in which death took place in a very short time, and this was the only lesion found. The subject has been more recently discussed by F. W. Draper, of Boston, who describes five cases (*Transactions of the Association of American Physicians*, vol. i.). The patient in

this case complained for several days of intense pain in the epigastrium, which was also very sensitive to pressure. He winced at the slightest touch, and it was thought possible, as he had lost flesh, and had a haggard appearance, that he might have a cancer of the stomach. There was nothing found to account for the pain, except the swollen state of the semilunar ganglia, and the condition of the pancreas above described. The mode of death was peculiar. He had taken his breakfast as usual, after a tolerably comfortable night, and at 9 o'clock was found by the ward attendant lying on his side, as if in a deep sleep, but breathing in a strange manner. When seen by the resident, Dr. F. A. Packard, he could be roused, but the pulse was almost imperceptible at the wrist, and there was typical Cheyne-Stokes respiration, with unusually long periods of apnœa. In this state he continued till 11.30, when death took place. The chambers of the heart, particularly those of the right side, were found to be dilated. The brain was normal. It has been suggested in these cases that the rapid death may be due to reflex inhibition of the heart, induced by the sudden shock to the solar plexus, which may be supposed to occur when hemorrhage takes place into the pancreas and adjacent tissues. It is analogous to Goltz's "Klopf-Versuch," in which a tap on the peritoneum of a frog inhibits the action of the heart.

Dr. Formad had seen, perhaps a dozen times, the pancreas full of blood. He remembered distinctly that in cases of arsenical poisoning it was often soaked with blood, also in cases of very acute alcoholic poisoning, when a quart or more of spirits is drunk, death often ensues without any other lesion than this. In a few cases of such poisoning the infiltration has been in spots and the organs presented a very peculiar appearance. *January* 28, 1886.

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6. *Sudden deaths from chronic gastric ulcer. Specimens of eighteen cases.*

Exhibited at various times within two years, by Dr. H. F. Formad.

None of these cases had been published anywhere previously. Dr. Formad had made all the autopsies, but had not seen any of these cases during life; and, unfortunately, had only imperfect information as to the symptoms or duration of the disease. It appears that none of these eighteen cases of gastric ulcer here recorded had been diagnosticated during life. In two or three cases in which the histories were more or less satisfactorily elicited, it could not be ascertained that the deceased had been aware of any gastric trouble during life, nor that there had been any vomiting except shortly before death. In only four of these cases was

there any protracted illness noted to precede death. Some of the cases had not been under medical attendance, and were thus referred to the Coroner in order to obtain a death certificate; while others that were under medical treatment had been submitted to legal inquiry on account of sudden or unexpected death; acute poisoning, criminal abortion, or some sort of foul play having been suspected.

Although unsatisfactory from a clinical standpoint, the cases here recorded present a series of quite peculiar character and worthy of study, offering points of interest pathological and statistical.

Dr. Formad believed that gastric ulcer was sometimes overlooked at autopsies, and was sure that he had done so himself. Gastric ulcer does not appear to be more rare in this country than in Europe, as has been alleged by writers. In the examination of the stomach the large curvature should be cut open, and not the small one, as he had seen some do. The rule must be observed, laid down first by Virchow, that gastric ulcers (and all chronic lesions) occur more frequently at and near the small curvature than at the large one. In making the incision through the lesser curvature of the stomach (which is often more convenient) small gastric ulcers may be cut through and escape notice.

In the series here recorded, Dr. Formad did not include any cases of mere scars in the stomach, or of multiple hemorrhagic erosions of acute character, or any lesions that are the result of injuries, corrosive poisoning, violent vomiting, etc. Occasionally gastric ulcers were found at autopsies in cases where they were least suspected, and often it was difficult to account for their occurrence; as will be seen in some of the cases described below.

It might be of interest from a statistical standpoint, that the eighteen cases of Dr. Formad's here recorded occurred in a total number of close to twenty-seven hundred autopsies made by him in the period stated above. This would correspond to one case in one hundred and fifty autopsies; but considering that nearly one-half of this total number was made up of cases of injuries, violence, suicides, and children's diseases, it is evident that the percentage of the occurrence of gastric ulcer in cases of sudden death may be put at one and a third per cent.

Dr. Formad did not include in these records any cases of gastric ulcer that he had seen (about ten or twelve) in his hospital and private autopsies, as these might have been, or will be, recorded by some one else.

Gastric ulcer among deaths from all causes in private and hospital adult cases, Dr. Formad thought, may be safely estimated at two per cent.

In order to facilitate the study of the cases here recorded, they might be conveniently divided into four classes, according to the mode of death:

Class I. Cases in which fatal perforation of gastric ulcer occurred.

Class II. Cases with perforation of gastric ulcer, but fatal termination being delayed by temporary healing and adhesions.

Class III. Cases without perforation of ulcer, but death occurring from gastric hemorrhage.

Class IV. Cases without perforation of ulcer, and death occurring from causes other than gastric ulcer.

Class I. Gastric ulcer, perforating into peritoneal cavity; death from rapidly fatal peritonitis. Six cases.

Case I.—I. J., female, white, æt. 29 years; married; well nourished. Said to have been of temperate habits; dyspeptic. Died of peritonitis; probable time about twelve hours after rupture of perforating gastric ulcer. Stomach overloaded with food. Single ulcer, round, half inch in diameter, terrace-like, sloping, sharp-cut edges. Location: slightly anteriorly from lesser curvature, near (about three inches) from pylorus. Chronic gastritis with dilatation of stomach. Mitral endocarditis with stenosis. Cyanotic induration of kidneys and spleen. Peritonitis.[1]

Case II.—N. O., female, white, æt. 33 years; single. Said to have been quite intemperate. Died suddenly from supposed poisoning. Perforation into peritoneum of a single, oval, three-quarters inch sized, gastric ulcer, located in lesser curvature, slightly posterior and near pylorus. Chronic gastritis with hemorrhagic infiltration; large fatty liver. Cyanosis of kidney and spleen. Peritoneum filled with liquid escaped from stomach, and showing signs of acute peritonitis.

Case III.—A. S., female, white, æt. 50 years; married; ill-nourished, lean body. Said to have been a sufferer from dyspepsia and gastralgia for years. Death from peritonitis due to perforation of large ulcer of lesser curvature near cardia, and escape of contents of stomach into peritoneum, probably about forty-eight hours previously. Size of ulcer one inch in length and three-quarters of an inch broad; edges swollen and indurated; much raised over level of mucous membrane, which apparently is substituted by scar tissue for some distance around ulcer. Hypertrophy of heart. Cirrhosis of all organs with fatty infiltration of liver superimposed. Uterine fibroids. Peritonitis.

Case IV.—H. D., female, white, æt. 30 years; single; of hysterical and dyspeptic habits; well nourished; a society belle. Died six hours (probable time) after the rupture of a perforating gastric ulcer. Autopsy showed two symmetrical and oppositely disposed gastric ulcers in the

[1] Any organs not referred to in this or in any of the following records, are meant to have been either in a visibly normal condition or in such states as could not possibly have had any connection with the gastric ulcer.

large curvature, at the fundus, and about one and a half inches on each side of median line. Size of each, a half to three-quarters inch; round shaped, sloping sharp-cut edges. Rupture of anterior ulcer. The posterior ulcer had only the eroded peritoneum as its bottom. Mucous membrane cloudy, swollen, but not mammillated or thickened. Mitral endocarditis; acute oöphoritis; intramural fibroid (size of large orange); evidences of former attacks of cellulitis and perityphlitis. Some liquid from the stomach had escaped into the abdominal cavity, and in spite of the short duration of but a few hours, a well-marked, fatal, acute peritonitis had established itself. This form of symmetrical gastric ulcers is very rare.

Case V.—B. D., female, white, æt. 40 years; married; hard drinker; body lean; found dead in bed. Peritoneum filled with contents of stomach that had escaped from a large perforated gastric ulcer; and the abdominal viscera matted together by a mixture of food, alcoholic liquids, and purulent inflammatory lymph. Location of ulcer near lesser curvature, posterior, and midway between cardia and pylorus. A second shallow gastric ulcer, round like the first and a half inch in diameter, in close proximity to perforation. Chronic gastritis; large fatty infiltrated liver; fatty and contracted kidneys with cysts; pleurisy. Apices of congested lungs indurated and cicatrized.

Case VI.—M. C., female, white, æt. 27 years; single. Hysterical; body quite emaciated; marks of syphilis. Death from acute cholera morbus. Chronic ulcer near pylorus, anteriorly from lesser curvature; single, sharp-cut, oval, one inch in longest diameter. Perforation into peritoneal cavity, probably, during an attack of vomiting. Mucous membrane of stomach showed numerous small hemorrhagic infiltrations and erosions. Poisoning suspected, but not substantiated by chemical analysis.

Class II. Gastric ulcer, perforating, but forming adhesions with liver (one case), and with pancreas (two cases); death from perforation. Three cases.

Case VII.—A. M., male, white, æt. 38 years. Occupation shoemaker. Body tall, much emaciated. "Had consumption and dyspepsia for a long while," is all that could be obtained as a history of the case. Marks of syphilis were, however, evident.

Autopsy.—Metastatic abscesses in both lungs and in anterior mediastinum; empyæma; atheroma of aorta with simple hypertrophy of heart; large ante-mortem clots in all heart-chambers. Liver showed a large multilocular abscess, chiefly of right lobe, containing nearly one quart of pus. Posterior wall of stomach, near lesser curvature and at its

middle, showed adhesions with left lobe of liver, and on opening, a large perforating gastric ulcer was seen, corresponding to the site of the adhesion and opening into, and in direct communication with the abscess of the liver. Perihepatitis. Fatty and contracted kidneys. Acute peritonitis. Pyæmia.

Case VIII.—E. P., female, white, æt. 29 years; domestic; single. Said to have suffered often from gastralgia and hæmatemesis. Body much emaciated. Gastric ulcer about three-quarters of an inch in diameter; round, terrace-like edged; location, lesser curvature posteriorly near pylorus. Ulcer perforated, but perforated part of stomach was in intimate union with the head of the pancreas by means of old adhesions made up of dense connective tissue. Pyloric end of stomach much thickened, and its mucous membrane quite mammillated; rest of stomach extremely dilated and filled with a brown liquid (microscope showed some blood). No acute signs of peritonitis. Other notes omitted at the autopsy, but I remember that there was valvular heart disease in this case.

Case IX.—K. F., female, æt. 36 years; German; cook; married; had six children; moderate drunkard. Died suddenly, about twenty hours after the probable time of rupture of a perforating gastric ulcer, during a coughing spell, it was said. Two gastric ulcers, sharp, round, about the size of a dime each, and in close proximity to one another, in posterior wall and about the middle of stomach. There had been some adhesion between bottom of ulcer and the pancreas—in fact, the latter had been forming the temporary floor of the perforating ulcer (the one nearest to fundus of stomach); thickened, raised edges, much hemorrhagic infiltration of mammillated mucosa; loosening of pancreas from the ulcer. Escape of food, solid and liquid, in large quantity into peritoneum on account of the tearing loose of pancreas. Acute suppurative peritonitis. Large fatty infiltrated liver. Pulmonary phthisis.

Class III. Gastric ulcer, not perforating; death from fatal hemorrhage into stomach. Two cases.

Case X.—J. H., female, white, æt. 40 years; single; body well-nourished. Said to have been subject to dyspepsia and gastralgia for about one year. Was a melancholy and partly demented individual. Died suddenly without having been under regular medical attendance, from a massive hemorrhage into the stomach. On opening the stomach showed a complete cast of coagulated blood. Examination revealed the source of the hemorrhage to be a gastric ulcer about middle of lesser curvature, somewhat posteriorly. Size, one inch in diameter. Shape, round, sloping, sharp, indurated edges; not perforated. Chronic mam-

millated gastritis. All organs anæmic. Some chronic endocarditis with stenosis of aortic and mitral valves. Hypertrophy of heart, cyanosis of kidneys, and atrophy of liver.

Case XI.—P. Mc., male, white, æt. 38 years; saloon-keeper; fat and strong; drunkard for years. Died suddenly after a few days of bad dyspepsia. Death from a rapid massive hemorrhage into the stomach, preceded for a few moments by violent hæmatemesis. Stomach filled to the utmost with a large blood coagulum that retained the perfect shape of the stomach after opening the latter. Ulcer in posterior wall of stomach, one-third of an inch in diameter, oval, funnel-shaped, extending only through mucous and muscular coat without perforating. Chronic gastritis, cyanosis of spleen and kidneys, and large fatty liver.

Class IV. Gastric ulcer not perforating; death from causes not dependent upon gastric ulcer. Seven cases.

Case XII.—A. F. C., female, æt. about 45 years; German; had children; well nourished; skin somewhat jaundiced; intemperate habits. Said to have complained often of "severe cramps in stomach and liver." Gastric ulcer in posterior wall near pylorus; shape, round; size, three-quarters of an inch in the thickened mucous coat, half inch in diameter in muscular coat beneath; serous coat intact. Stomach dilated and containing dark blood-stained viscid liquid, evidently due to slow hemorrhage from the ulcer. (Clothing of deceased indicated, also, that she had been vomiting blood shortly before death.) Liver fatty infiltrated. Kidneys cyanotic. Heart dilated, flabby, with large antemortem clot in left chambers; chronic endocarditis with calcification of aortic valve; atheroma of coronory vessels and of aorta. Death was due to heart failure.

Case XIII.—G. A., male, white, æt. about 40 years. Died from effects of acute alcoholism. No reliable history could be elicited. Chronic gastritis and great hypertrophy of muscular coat of pyloric region of stomach; hypertrophy of heart; brown induration of lung; œdema of brain; cyanotic induration of spleen and kidneys, the latter large, "pig-backed." When stomach was subsequently well washed, and more closely examined by an assistant, a gastric ulcer partly cicatrized was discovered. Size, nearly one and a half inches in longest diameter, heart-shaped, edges smooth. Serous coat at bottom of ulcer intact and quite thickened. Location, small curvature and anteriorly in pyloric region.

Case XIV.—M. McC., female, white, æt. 26 years; single; had no children. Died in syncope. The case had been supposed to be one of criminal abortion. Uterus showed an intra-mural fibroid that had led to fatal bleeding. Uterus and ovaries appeared to show signs of men-

struation. Chronic gastric ulcer three-quarters of an inch in diameter, sharp-edged and round; no perforation. Location, posteriorly near small curvature, middle of stomach. All organs extremely anæmic.

Case XV.—A. Y., female, white, æt. 45 years; demented; had children years ago. Died during an alcoholic debauch. Two gastric ulcers partly filled with cicatrizing granulations. Size of one ulcer nearly two square inches; size of second ulcer three-quarters of an inch in diameter. Both ulcers irregularly shaped with raised indurated edges, and located closely together in anterior wall, about the middle and near lesser curvature of stomach. Chronic gastritis with mammillation of mucous membrane, and showing also numerous minute hemorrhagic erosions. Large fatty infiltrated liver, with biliary infarcts. Gall-bladder contained twenty-two small, dark gall-stones. Large intestine showed several scars and much hypertrophy of mucous membrane. Kidneys, alcoholic cyanosis.

Case XVI.—Ann R., female, colored, æt. 90 years; single. Said to have been slightly demented for some time. She had lived for many years on liquid food and oysters. Habits temperate. For thirty-five years she had the same medical attendant; but gastric ulcer had not been suspected. Body weight about seventy pounds, of which eight pounds pertained to uterine fibroids. The whole body with its organs was greatly atrophied; no visible adipose tissue anywhere. Lungs anthracosed like those of a miner, otherwise normal as well as the heart. Intestines had a very narrow lumen with thickening of walls throughout, but showed nothing abnormal except some congestion in lower part of ileum. Stomach showed two large ulcers, located in the middle of small curvature, both posterior to the median line and close together. Size of each ulcer, one inch and a half long and one inch broad; irregularly oval in shape, edges quite rounded, indurated and elevated. The much thickened serous coat of the stomach formed the bottom of the ulcers; a compensatory development of connective tissue preventing the rupture of the ulcers. The mucous membrane of stomach (which was empty) was greatly atrophied and was deeply congested from an acute attack of gastritis. (The deceased had vomited repeatedly and complained of gastric pain only during the last day of life, so that the question of poisoning was raised, but was not substantiated by chemical analysis.) The appearance of the gastric ulcers suggested that the latter had been in existence for many years.

Case XVII.—N. P., male, white, a notorious miser, probable age about 60 years. Died from starvation and by freezing during a cold winter night, in an unheated room in one of the many houses that he owned in Philadelphia. Deceased had been living for many years on

scanty and the worst kinds of cold food, which he obtained gratuitously in taverns while taking alcoholic drinks, he being quite intemperate. No other history.

Autopsy made when body was discovered in locked room three or four days after death. Besides lesions peculiar to alcoholism and atheromatous bloodvessels, there was found a single, round, not perforated, gastric ulcer in smaller curvature near pylorus; size, half an inch in diameter. Appearance suggested that it was of long standing. There was, also, a large scar near the ulcer, posteriorly, indicating the healing of a former similar lesion.

Case XVIII.—M. M., female, white, æt. about 32 years; had children; highly intemperate. Killed accidentally by her husband, a tavern-keeper. Lesions peculiar to alcoholism found at the autopsy; also a single, large gastric ulcer, nearly an inch in diameter, with raised, rounded edges in the much thickened mucous membrane of small curvature of stomach, somewhat posteriorly and near pylorus. No perforation.

Remarks and Résumé.—An analysis of these cases of gastric ulcer shows: males, four cases; females, fourteen cases. All occurred in white persons but one (the colored woman, aged 90 years). Two were Germans, fifteen were of Anglo-Saxon extraction.

Chief location of ulcer in lesser curvature and always laterally; six being anterior and eleven posterior to the median line. In only one case were the ulcers located in larger curvature (Case IV.). The deceased had not been aware of any gastric trouble during life in most of these cases.

Age at time of death: between 26 and 30 years, 6 cases; between 30 and 40 years, 8 cases; between 40 and 50 years, 2 cases; between 50 and 60 years, 1 case; 90 years, 1 case.

In three instances the ulcers occurred in demented, two in hysterical women.[1] In only four cases was alcoholism denied, while in eight intemperate habits were a conspicuous feature; in six not known.

Valvular heart disease was met with in five cases; atheroma of aorta in two; phthisis in two; Bright's disease in two; liver abscess in one case.

In only one case could tight lacing and pressure by lower border of ribs be suggested as a causative factor; this was in Case IV., and here the location of the ulcers was in the large curvature of stomach.

The mode of death: Peritonitis and shock from perforation of ulcers in six cases.

[1] Dr. Francis Dercum has directed attention to the occurrence of gastric ulcer in the insane on one or two occasions before this Pathological Society.

Fatal termination by peritonitis after rupture of chronic adhesions to liver and pancreas, by means of which perforated gastric ulcers had been temporarily closed in three cases.

Fatal massive hemorrhage into stomach without perforation of gastric ulcer in two cases.

Fatal termination from other than gastric lesions coincident with and irrespective of the gastric ulcer in seven cases.

The histological details and some observations upon the etiology of gastric ulcer, will be treated of by Dr. Formad in a future paper.

February 25, 1886.

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7. *Gastric ulcer; abscesses of the liver.*

Exhibited by Dr. E. T Bruen.

Dr. Bruen reported a case of gastric ulcer situated near the line of the lesser curvature, in the middle zone of the posterior wall or the stomach. Adhesions had formed between the posterior wall of the stomach and the mesentery, and uniting the head of the pancreas to the inferior aspect of the liver. Within these adhesions an abscess had formed which on autopsy communicated with the stomach through rupture of the ulcer already described.

The liver was very much enlarged and contained numerous large abscesses. In fact, the entire organ was a series of abscesses—only a very small portion of hepatic tissue remained. The branches of the portal vein, as they divide after leaving the main trunk, were normal, as was the portal vein itself. The intestines were normal. The pus in the liver was yellow, laudable pus, no trace of chocolate tint, the abscess cavities possessed rugged walls, so that the tissues were riddled with the pus. Perihepatitis with exudation of lymph had existed, and some pleurisy on the right side at the base and over the lower lobe. The exudation from above processes was lymph—but little or no pus. The upper lobe of the left lung was the seat of fibroid induration; but, in addition, a vomica as large as a lemon and filled with pus occupied the upper part of the apex. The patient was a negro, a male, about thirty years of age, fairly nourished, an hostler by calling, and in spite of the latent phthisis had not considered himself ill till about eight weeks before his death, and the increment of his symptoms was very gradual. During his illness there was never any complaint of pain over the liver or epigastrium—no sweating, no jaundice, no pigmented urine. There had been no vomiting or diarrhœa, It was manifest, during life, that the disease

was some process in the zone of the body below the diaphragm, the evidence being chiefly by exclusion. The typhoid state was present, and the patient gave the history of a prodromic period with nose-bleed and diarrhœa, which suggested typhoid fever. The temperature ranged between 103° and 104° F., highest in the evening, for two weeks, which gave color to the hypothesis. The duration of the case rendered the diagnosis untenable. During the last two weeks wide ranges in the temperature curve were observed, for instance, from 97° to 106° F., with occasional chills. The liver was also found to be enlarged, yet the diagnosis was subject to debate when death occurred.

The most characteristic symptom of hepatic suppuration was the temperature, especially the subnormal records, without sweating, which is often absent in hepatic abscess, *vide* Carrington in the *Guy's Hospital Reports* for 1884. Yet Ughelti, in the *London Medical Record* of June 15, 1885, cites a case of Ciaramelli's without fever, in which a vast abscess of the liver was found encapsuled by a thick layer of connective tissue which prevented the reabsorption of pus.

In regard to the ulcer of the stomach, it is of interest to note the absence of special symptoms significant of the lesion, that it occurred in a well-nourished male, but in one who probably used alcohol to excess at times. The location is the usual one (*vide* Rokitansky), but the presence of the encysted abscess, the perihepatitis without general peritoneal involvement, and without the history of pain or abdominal distention, was truly remarkable. The patient, during his seven weeks' illness, always expressed himself as feeling fairly well. Certainly, in this case, the connection between the ulcer of the stomach and the hepatic abscess must be inferential, though no signs of phlebitis were detected.

February 25, 1886.

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8. *Tubercular ulceration of the ileum.*

Exhibited by Dr. F. P. Henry.

R. H., married, æt. 50 years. A good family history, both parents living and healthy. He was a periodical drinker, his debauches lasting from two to three weeks, and occurring generally about twice a year. He twice attempted to commit suicide while drunk, once with arsenic and once with Paris green. The last attempt was made in February, 1882. He began to fail in health during the summer of 1885, but worked until the middle of October. He then had severe pain in the region of the

kidneys, and was prescribed for by his family physician, Dr. D. W. Bortz. This pain left him, but then he began to get short of breath on exertion, and gradually lost flesh. He complained of an uneasy feeling in the epigastrium. I saw him first, December 24, 1885; he then was feeble and short of breath on the least exertion. Had a dry cough, and often in the morning on rising vomited mucus. He had no pain, hemorrhage, or anasarca. Could sleep well; ate moderately well; never vomited food. Heart action rapid and feeble. He complained of nothing but the distress in his epigastrium, which did not amount to pain. Bowels regular, passage once a day. Saw him again, January 16, 1886; had lost considerable strength since my last visit; cough unabated. Urine examined, no albumen found. Still no hemorrhage, anasarca, or pain. Slight redness in right iliac region; abdomen slightly tympanitic in this region. Saw him again, February 3d. Cough allayed; vomiting of mucus stopped. He is very much weaker and gets very short of breath on the slightest exertion. Has very little thirst; no appetite, whatever, for some days. Muscles rigid over stomach, but no redness. Abdomen slightly tympanitic all over. No pain anywhere; no œdema of feet; no hemorrhage. He died of exhaustion, February 6, 1886.

Autopsy.—Body emaciated. No serous infiltration. Slight amount of serous fluid in pericardium. Heart normal. Liver substance brittle and somewhat atrophied. Degeneration of mucous membrane of stomach throughout the cardiac half. Stomach contained a greenish offensive fluid. Left suprarenal capsule contained a solid mass. Slight solidification at apex of left lung. Five transverse ulcerations in the ileum, about three-quarters of an inch wide. *February* 25, 1886.

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9. *Amyloid degeneration of the liver.*

Exhibited by Dr. H. M. Fisher.

Margaret J., age unknown, apparently about 40 years, colored, was admitted to the Episcopal Hospital, September 9, 1885. For the following notes of the case I am indebted to Dr. C. J. Irvin, the Resident Physician. The patient was a widow, had never had children, and apparently no miscarriages. She had had no serious illness, but had suffered from rheumatism. The present attack began with cough and shortness of breath and palpitation of the heart. Her feet and legs then

became swollen, and in March, 1885, she was obliged to stop work. In April her abdomen began to swell and her cough ceased, but her other symptoms continued. Soon after admission to the hospital her dyspnœa became so great that it was found necessary to tap her. It was noted at that time, that there was a good deal of gastric disturbance, vomiting continuing for two or three days at a time, and with difficulty relieved. Her hands and face had been somewhat œdematous for two or three weeks before admission. The apex beat of the heart was in the third interspace, just inside of the nipple line. She had frequent attacks of severe and almost uncontrollable vomiting, and during the last two months took almost nothing but cocaine hydrochlor. gr. ss t. i. d., almost all other remedies that had been tried having occasioned nausea. She had, on one occasion, diarrhœa for several days, that was checked with difficulty, and her abdomen was constantly very sensitive to pressure. In the intervals of nausea her appetite was very good. She was tapped eight times during her stay in the hospital, and very nearly seventeen gallons of fluid were removed from her peritoneal cavity during these six months. Her pulse, which was feeble and intermittent when I first saw her, toward the end of November became more and more feeble. She finally died of exhaustion, March 6, 1886. I omitted to state that her urine was albuminous and was found to contain casts.

Autopsy, seventeen hours after death.—Body very much emaciated. Recent lymph on surface of small intestine, extending to transverse colon. Transverse colon firmly adherent to omentum. Upper surface of liver covered with a layer of organized lymph one-twelfth of an inch in thickness. There were several cicatrix-like excavations in this lymph deposit. Liver weighed three pounds one ounce; there was intense congestion of its lower peritoneal investment; its cut surface was markedly translucent and waxy in appearance; its lobes were tightly adherent from inflammatory exudation. There was a small collection of cheesy matter, about the size of an almond, at the upper third of the right lobe, and other collections of a like kind in the immediate vicinity. On the addition of iodine a reddish-brown coloration appeared. The mucous membrane of the stomach was intensely congested. The pancreas was abnormally hard in texture and hyperæmic. The kidneys were contracted and fatty. The spleen, through some accident, was not examined. The intestines do not appear to be in any point very markedly congested. The lower lobe of the left lung was very markedly œdematous, and there was much congestion of the right lung. Heart flabby.

The principal interest of this case rests in the causation of the amyloid infiltration. No history of suppuration was elicited, nor did I learn that

there was any history of syphilitic infection. The small cheesy foci, however, may be due to the softening of small gummata, and the enormously thickened capsule of the liver makes the case bear a strong resemblance to some cases of syphilitic disease.

March 11, 1886.

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10. *Tumor of the stomach.*

Exhibited by Dr. J. M. Barton.

W. McL. was sent to me in April, 1885, for the removal, if I thought advisable, of what appeared to be a cancer of the pylorus. The patient, æt. 47 years, had been drinking heavily from the age of forty to forty-five, when he began to suffer with digestive troubles. In November, 1883, he first noticed the tumor, and in January, 1884, he had a severe hemorrhage from the stomach. He now (April, 1885) complained of a constant burning sensation in the epigastrium, increased immediately and greatly by food, and causing immediate nausea and vomiting, the vomited matters consisting of the food swallowed, mucus, and occasionally bile. For the last seven weeks he had not been able to retain anything whatever on his stomach and had lost altogether forty-three pounds in weight. He had a hard, well-defined swelling in the epigastric region about two by three inches in its diameters and just below where the pyloric extremity of the stomach should be; tumors here generally descend by gravity during their growth, carrying this portion of the stomach with them, as pointed out by Prof. Tyson. As the symptoms were not those of pyloric obstruction, but evidently due to a chronic gastritis present, I did not regard the case as a suitable one for operation, without regard to the malignancy or non-malignancy of the tumor. As the patient was very urgent for operative interference, I gave him the opportunity to consult several of my medical friends in whom I had confidence, and among whom there was quite a difference in opinion regarding the character of the growth. Under medical treatment the symptoms greatly improved, he returned to work and increased greatly in weight. The patient recently perished from acute purulent pleuritis and pericarditis. The tumor occupied the upper portion of the pyloric outlet of the stomach and did not encroach enough upon the outlet to prevent my finger readily passing through. The tumor was smaller than I expected to find it; was hard, and was not ulcerated. Dr. de Schweinitz and Dr. Formad made a microscopical examination of the growth and found it to be scirrhous carcinoma.

April 8, 1886.

11. *Soft carcinoma of intestine; profuse metastasis; obstruction-pneumonia, and lesions of former hemiplegia in the same case.*

Exhibited by Dr. T. S. K. Morton.

Man, æt. 44 years, admitted with a high temperature, diarrhœa, and beginning pneumonia of right side. Had post-hemiplegic chorea of left side of face. Pneumonia became well marked. Symptoms as above, together with quite high temperature, continued for nine days, when he died of exhaustion. Two days before death, symptoms of pressure upon the trachea, œsophagus, and sympathetic nerve of the right side were noticed, and gave rise to the suspicion of aneurism by Dr. Hutchinson, in whose wards the case was.

Post-mortem were found very interesting lesions of old hemiplegia in the middle and anterior lobes of the right brain; a girdling soft cancer of the jejunum, which involved the neighboring mesentery, but not materially the intestinal lumen; metastases of varying size to spleen, liver, kidneys, stomach, and lungs. This last affected the bronchial glands of the right side, and involved all surrounding structures, including lung, œsophagus, pericardium, wall of auricles, and totally occluded the right bronchus. The pneumonia was due, probably, to this obstruction, and, therefore, was of the type known as obstructive pneumonia, or pneumonia of obstruction. *May* 13, 1886.

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12. *Spleen from a case of typhoid fever dying of septicæmia; showing puriform degeneration of old infarcts.*

Exhibited by Dr. T. S. K. Morton.

T. N. could remember no sickness to which he had been subject previous to the one for the relief of which he applied for admission to the Pennsylvania Hospital. Seven days previous to this he began to feel drowsy, had several chills, much headache, and high fever, also profuse diarrhœa; which, indeed, together with dicrotic pulse, full and tender abdomen, congested lungs, and a dry, brown tongue, were his symptoms upon admission. The spleen was found to be somewhat enlarged, though partially obscured by tympany. Urine normal. He lingered thirteen days, and then died. During this period, constant and uncontrollable

hyperpyrexia, diarrhœa, tremendous sweating, delirium, and occasional cyanosis to lividity were the rule. Once or twice, when the cyanosis occurred, he seemed much shocked, and the temperature fell several degrees, but almost immediately rose to 104°–105°. He complained much of pain in the splenic region several days before death.

Post-mortem examination revealed the usual manifestations of typhoid fever in the third week, together with congested lungs, signs of old slight endocarditis, and the condition of spleen about to be described.

The organ weighed almost two pounds. Throughout its entire extent it was riddled with inflammatory sacs, varying in size from that of a marble to a hen's egg, and larger. Each of them represented an old and broken-down hemorrhagic infarct, or coalescence thereof. Their contents varied in composition. Some, usually the smaller ones, were filled with inspissated and decolorized blood and inflammatory products, or preserved the usual structure of such infarcts; while in others, this material had undergone puriform degeneration, and thus formed abscesses, or, more properly, perhaps, residual abscesses. As far as the above lesions are concerned, the man's medical history is probably as follows: Many years before the last illness he had endocarditis, with extensive splenic infarction, and then acquiring typhoid fever, and his vitality being heavily drawn upon, these infarcts, or their products underwent purulent degeneration, were absorbed in part, and he died of septicæmia. *May* 13, 1886.

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13. *Abscess of liver.*

Exhibited by Dr. J. C. Wilson.

A. M., æt. 57 years, colored.

Autopsy.—No external marks, except abdomen decidedly distended. Body extremely emaciated.

Thorax: Bilateral pleurisy in slight degree. Some adhesions and effusion. Both lungs much compressed, particularly the right one, from pressure exerted by the enlarged liver, the diaphragm being pushed up to the level of the fourth rib on the right side. The right lung formed firm adhesions at the base with the diaphragm. Lungs were somewhat atelectatic and congested, otherwise normal.

Heart: Small in size—showed attenuation of muscular walls from atrophy. Very pronounced atheroma of the coronary vessels, but no valvular lesions. Ante-mortem clots on both sides.

Pericardium contained a considerable amount of clear liquid. Aorta slightly atheromatous in its ascending portion.

Abdomen: Liver appeared enlarged to nearly double its normal size, particularly the right lobe; but upon examination it was found that this enlargement was due to two large abscesses located side by side in the anterior part, and involving the greater portion of the right lobe of the liver. Each of the abscesses was the size of a newborn child's head, and was overdistended by a mixture of pus, and partly gelatinoid or myxomatous material. Floating in this liquid were masses of liver tissue, and fragments of a substance resembling chicken-fat clots; also, some fragments of red blood-clots. The wall of these abscesses appeared to be chiefly formed by the capsule of the liver, and the partition between the two abscesses, which were close together, did not exceed one-half inch in thickness. The inner lining of the abscesses, after removal of the pus, appeared like the surface of a large ulcer, with numerous bands of connective tissue threads hanging from it. The rest of the liver appeared in a state of fatty infiltration, and had a pale, nutmeg appearance. The surface of that part of the liver which was intact was smooth.

The gall-bladder and its ducts, as well as the portal vein, did not show any abnormal condition.

Spleen was atrophied, as well as kidneys, without presenting anything abnormal.

Stomach was in a state of chronic gastritis, showing thickening.

Small intestine was in a state of catarrhal inflammation, with some enlargement of follicles.

Large intestine gave a typical appearance of chronic catarrhal or follicular dysentery, with great thickening of mucous membrane, which was riddled by indurated ulcers of various sizes, from a pin's head to a line in diameter, many showing a loss of substance. *May* 27, 1886.

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14. *Simple dilatation of the large intestine.*

Exhibited by Dr. W. E. Hughes.

The patient, a boy, æt. 3 years, was troubled, in common with many other children in early infancy, with obstinate constipation. At birth the size of his belly was normal, but as he grew older and the constipation more marked, it gradually enlarged. The passages, when he did

have them, which was only after the exhibition of laxatives, were normal; they were never hard. An injudicious persistence in the use of rectal enemata seems rather to have aggravated the condition. In spite of the constipation he throve and grew and digested his food well till he was about two years old. Then, in the summer, he had a sharp attack of entero-colitis. After this his digestion was not good and his health was poor. The constipation became much more obstinate—long periods elapsing between the ejections of fecal matter. At one time he went nineteen days without a passage. The longest of these periods were attended with uneasiness, restlessness, and marked loss of appetite and digestive power, but never pain or vomiting. In this condition enemata seemed to do little good, and it was only after the persistent use of rather strong purgatives that an evacuation of the bowels could be obtained. The use of large enemata was, however, steadily continued. When first seen he was a poorly nourished child with an evident expression of anxiety. The belly was symmetrically and enormously enlarged and everywhere tympanitic, with enlarged veins coursing over its surface. Incessantly waves of peristalsis rolled along the intestines, outlining them sharply. These waves were plainly in the colon, which was evidently enormously distended. They were often attended with much pain to the child. This time it was twenty-four days before an evacuation of the bowels could be secured. After a period of apparent steady improvement, the child died of an acute attack of colitis.

Autopsy.—There was nothing remarkable except the colon. It was enormously dilated, and held, when taken out, fourteen pints of water. The greatest dilatation was in the upper part of the rectum and lower part of the colon, where it was four inches in diameter. The muscular part of the wall was enormously hypertrophied, growing progressively thicker from the cæcum to the anus. The mucous membrane was thickened and contained numerous small, shallow, rounded ulcers. There were no cicatrices, nor any evidences of an old volvulus. About the middle of the transverse colon was a large area of recent inflammation. Both old and recent inflammations were much the most marked opposite the longitudinal muscular bands.

This case is an interesting one as helping to illustrate the tendency of all the hollow viscera to dilate under any abnormal distending force, coupled, perhaps, with weakening of the walls. Its etiology is evidently identical with that of dilatation of the stomach. It shows, too, that even with weakness of the walls, there will be hypertrophy consequent to the dilatation, vainly attempting to recover lost ground. A minor degree of distention of the colon probably attends any moderate attack of entero-

colitis; that this dilatation eventually returns to the normal is probably due to the fact that the colon can so easily empty itself. Clinically, it teaches the worse than uselessness of any continued use of large enemata, for these only add to the trouble by distending an already over-distended gut. *May* 27, 1886.

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15. *Cancer of the stomach.*

Exhibited by Dr. J. H. Musser.

H. H., æt. 59 years; white; widow; domestic; native of Ireland. Was admitted to the Woman's Medical Ward of the Philadelphia Hospital, July 30, 1886. Family history unknown. Has had rheumatism and malaria. Symptoms of present illness began December 25, 1885, with vomiting. The vomiting continued until admission, and was attended with some pain and gradual emaciation. The matter vomited was simply the food. The vomiting occurred twice or thrice daily, sometimes before, again after eating.

On admission the patient was slightly emaciated and presented a cachectic appearance, with anæmia. Appetite lost; vomiting one or two hours after eating occurred daily; obstinate constipation was present; also, more or less constant pain in the epigastrium. On examination the epigastrium was tender on palpation; pulsation was seen and felt, but no thrill detected; in the area above the umbilicus, a clearly defined hard mass, slightly movable, painless, the margins of which shaded off into ill-defined indurations in the hypochondria and epigastrium, was readily detected. The tumor appeared to be the size of the fist, and dulness was elicited on percussion. The abdomen was not distended; the walls were relaxed. Liver dulness was not increased.

Death took place on the 27th of October. During the time she was under observation the following symptoms and appearances were present:

1. Emaciation, progressive and extreme. 2. Cachexia. 3. Anæmia, with reduction of red cells and hæmoglobin. 4 Anorexia. 5. Vomiting of food and of a brown, frothy fluid, in which blood-corpuscles were detected but no sarcinæ. 6. Tumor; this did not increase in size, but its connection with the induration in the epigastrium became more pronounced; it became more tender.

No marked hemorrhages occurred; no secondary lymphatic involvement was observed. Duration of symptoms, ten months; length of time unable to work, four months.

Clinical diagnosis.—Carcinoma of the pylorus.

Autopsy, fifty hours after death.—No rigor mortis; marked emaciation; appearance anæmic and cachectic; slight œdema of ankles; lungs and heart normal; brain not examined.

On section, the mass situated in the median line extending from one inch below the xiphoid cartilage to the umbilicus, was composed of the pyloric end of the stomach attached to the left lobe of the liver. In the left lobe of the liver in the median line, a small, soft, white mass, attached to the pylorus as above, was seen. This portion of nodule in the liver was larger than a silver dollar, and was directly in the median line. The portion made up of diseased pyloric orifice was two inches in width, and extended to the right of the median line about one and a half inches. On removal of the stomach, the cancer in a state of ulceration was found to involve the pyloric end of stomach, pylorus, and two inches of the duodenum. There was no absolute stricture. The latter portion was adherent to the left lobe of the liver, where there was a large nodule with slightly raised umbilicated surface with a fluctuating centre, the size of a hen's egg. On section, the central portion of the liver node was found softened, and communicated by a sinus with the diseased pylorus. The gall-bladder contained fluid and a gall-stone the size of a pigeon's egg; the liver was in a state of fatty infiltration; the kidney and spleen normal; the thoracic organs normal; the heart contained a large ante-mortem clot; no effusion in pericardial or pleural cavities.

May 27, 1886.

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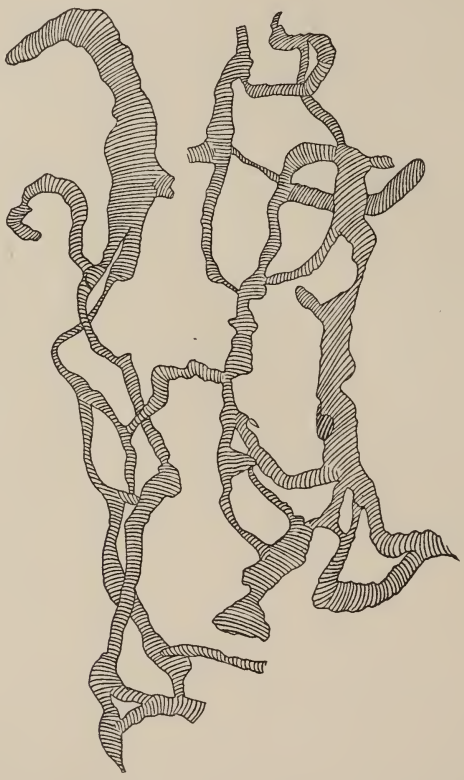
16. *Case of tubular diarrhœa.*

Exhibited by Dr. J. P. C. Griffith.

The specimen of so-called intestinal cast which I exhibit this evening, was passed by Mrs. S., æt. about 65 years, who had suffered for years from rheumatoid arthritis. For several days she had had some tenesmus with slight diarrhœa. I was shown what had been passed on August 27, 1886, and found it to consist of a whitish, thickly gelatinous material in a very small amount of clear, almost colorless fluid. The mass would have perhaps filled two tablespoons. On closer examination, it was seen to be composed of an irregular, intricate network of round, solid, white, tube-like cords of uneven, wavy outline, and varying in thickness from that of coarse thread to that of a lead-pencil; the general outline being uneven, as in the sketch which I exhibit. Some of the larger cords were streaked with a small amount of yellow fecal

matter. The meshes of the network varied in size and shape, and were nowhere greater than a half to one inch in length. There was no portion of the cord-like substance extending for a distance of more than one to one and a half inches without sending off one or more branches to unite with other portions of the network.



In consistence the substance was gelatinous, slippery, not bearing manipulation out of water, but sufficiently tenacious to retain its shape while in it. Shreds of whitish membrane of a nature similar to the cords, and a good deal of transparent mucus, were found with the network. Portions of the material were treated with various reagents, with the following results:

KHO dissolved it almost completely; HNO_3 dissolved it rapidly, with the evolution of abundant brown fumes; dilute HNO_3 softened it, and slowly and partially dissolved it; $C_2H_4O_2$ caused it to shrivel and become more dense.

Microscopical examination under a low power revealed no follicular appearance such as has been sometimes described. Under a greater amplification there were seen rather numerous small, round and spindle-shaped, lymphoid cells with a few epithelial elements imbedded in a hyaline, very slightly granular, mucoid matrix. There was but little fibrillation, and nothing resembling fibrin. Both microscopical and chemical examination prove that the matter is mucus.

It is not necessary to give a detailed clinical history of the disorder, as an exhaustive account of it with a bibliography, some of which is almost antiquarian, is to be found in the *Medical and Surgical History of the War of the Rebellion*, vol. ii., Medical Part. It may be said briefly, that it occurs oftenest in hysterical women, is preceded frequently by dyspeptic trouble, and membranous or tubular mucous masses are then expelled, accompanied sometimes by colicky pain. The fecal passages are usually more or less loose preceding or following the evacuation of the mucous material.

The disease is no new one, and I imagine, no unusual one. It probably occurs much more frequently than is suspected, since the subjective symptoms are often so slight. It is not in any sense identical with the "Mucus Disease" of Eustace Smith, and it is certainly distinct from dysentery, although its seat is probably in the colon. Dr. Osler informs me that he has found the mucous masses lying free in the colon at autopsies. It has been known since 1554, when Fernelius described the substance passed as inspissated mucus. I do not like the name "intestinal cast," as it in no way describes the appearance of the passages of such a case as the one reported to-night. This term might be applied more properly to some forms of dysentery. The title "Tubular Diarrhœa," first applied by Good, and preferred by Woodward to the very numerous synonyms, seems to me to be a very good one, as it implies no theory whatever. At times the mucus is passed in the form of membranes instead of cords, and in such a case we might, as Woodward says, adopt the term "Membranous Diarrhœa," although the affection is the same. Perhaps "Mucous Colitis" would describe both forms of the disease. *September* 23, 1886.

17. *Gummata of the liver.*

Exhibited by Dr. H. F. Formad.

The patient from whom this liver was obtained, was a woman who presented many of the lesions of syphilis. Upon the genitals were both open sores and old cicatrices. Upon the right side of the head was a large tumor fully one inch in diameter, with an open ulcer at its summit. Beneath it the bone was perfect, with the periosteum possibly a little eroded. Immediately beneath was a large gumma of the dura mater. Pressure by this tumor might possibly account for the disorder of vision which was present during life. The liver contained a series of gummata, the largest of which was three inches in diameter. What is especially interesting to me in this case, is the fact that, two months later I made an autopsy upon the body of this woman's child, six years old, and found in its lungs near the roots and bases, large gummatous masses. The other organs in the child presented nothing peculiar except that the lymph glands were enormously enlarged, and there was some catarrhal enteritis. *September* 23, 1886.

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18. *Stomach from a case of poisoning by hydrochloric acid.*

Exhibited by Dr. M. H. Fussell.

The described specimen was obtained from a case in the practice of my friend, Dr. A. H. Mellersh, of Roxborough, for whom the writer made the post-mortem examination.

M., male, æt. 45 years, of dissolute habits; had several attacks of delirium tremens. During a drinking bout he swallowed two fluidounces of laudanum with suicidal intent. Thinking that the poison was not acting rapidly enough he swallowed about one fluidounce of hydrochloric acid. When discovered the patient was vomiting profusely, with all the attendant symptoms of mineral poisoning. Curiously enough, however, there were no marks of the acid about the mouth or fauces. Almost an hour passed after swallowing the acid before Dr. Mellersh saw the case. Vomiting continued for a number of days, but was finally subdued by the use of opiates and demulcents. The patient then gradually improved, and it appeared as though he would recover; in a few weeks, however, vomiting returned. This time vomiting was generally delayed for some

hours after eating, when large amounts would be disgorged. Patient gradually failed and became extremely emaciated. Feeding by the rectum became a necessity. About this time an œsophageal bougie was passed, but the gullet was found perfectly free from stricture. Weeks after swallowing the acid death occurred.

Post-mortem examination.—Body extremely emaciated. No external marks. On section of abdomen the muscles were easily separated from the peritoneum, the latter being drawn very tightly between the iliac bones and binding the intestines to the posterior wall.

Lungs crepitant throughout—a small pleuritic adhesion at right apex.

Heart-muscle flabby and easily torn. Valves normal.

Liver and *spleen* normal.

Stomach small. On the anterior surface at the pyloric end was a depressed, slightly reddened spot, appearing like a partially healed ulcer. On filling the stomach with water from the cardiac end the water was retained by a narrowing just inside the pylorus. Water forced into the duodenum was prevented from reaching the pylorus by a narrowing of the gut just below the pylorus. When the gut and stomach were distended the whole gave a sort of double hour-glass appearance, the pylorus representing the middle bulb, the stomach and intestine the end bulbs, while the strictures represented the narrowed portions between the bulbs. On the cardiac end of the distended stomach were numerous prominences, ranging from the size of a hickory-nut to a pigeon's egg, giving the appearance as though the whole cardia was covered with bullæ. On slitting open the stomach the whole mucous membrane was seen to be destroyed, only cicatricial bands remaining. The remaining thinned coats of the stomach were distended between these cicatrical bands, and gave rise to the above appearance of the distended cardia. Over the seat of the scar near the pylorus above mentioned, the coats of the stomach were much thinned—almost as thin as paper. A small probe could be passed with difficulty from the gut into the stomach, folds of membrane causing the complete occlusion. On laying open the strictures the portion in the stomach was one inch by three-eighths of an inch, that in the duodenum, one inch by seven-eighths of an inch. The mucous membrane of the duodenum below the stricture was normal. The opening of the bile-duct into the gut was patulous.

November 11, 1886.

19. *Abscess of the liver.*

Exhibited by Dr. M. Rively, for Dr. D. D. Stewart.

Annie B., white, æt. 3 years, 11 months, 25 days, died October 17, 1886. She had been healthy, with the exception of an attack of measles when nine months old, up to her last illness; was well nourished; family history excellent. About the middle of last July—three months previous to death—while running along the street she was thrown violently down, her chest and abdomen coming with great force against the pavement. She complained for a day or two after of some soreness about the chest and abdomen, then the matter was forgotten. About ten days subsequent to the fall she again complained of the soreness, which had now assumed the character of a dull ache, and was referred by her to the epigastrium. This dull, aching pain was apparently continuous, with exacerbations most violent in character (judging from the manner in which she comported herself at these periods) occurring at intervals of an hour, and lasting a minute or two. She would refer these acute pains to the umbilical, epigastric, and right hypochondriac regions. But a proper understanding of her sensations was impossible, both on account of her age and the paroxysms of violent screaming she was thrown into whenever approached or touched by anyone. At the same time that these pains appeared, the mother noticed the child would complain at intervals through the day of chilliness, and her feet and hands would assume an icy coldness. The chilly sensations were followed in the course of an hour or so by a severe fever; and the fever, after a few hours, by perspiration. After a week from its onset, the paroxysmal pain entirely disappeared, leaving, however, a continuous feeling of discomfort. The seat of this uneasiness she could not locate when asked where she felt bad, letting her hand glide from the epigastrium to the umbilicus, or to various portions of the back, scarcely ever placing her hand twice in the same situation. The mother did not remember that she complained of pain in the right shoulder. She was at no time during this period confined to her bed, but was about each day, and ate fairly well. Her tongue was noticed to be quite heavily furred, and she had apparently lost a good deal of flesh. She had occasional attacks of vomiting, but they occurred nearly always after the administration of medicine, and never spontaneously or by food. The mother did not notice any yellowness of the skin or eyes at any time. After two or three weeks from the onset of the daily attacks of chilliness, fever, and

sweat, she ceased to complain of feeling cold, and her mother fancies her fever became continuous.

Dr. Stewart was called to see her a few days before her death on account of a bad cold she had contracted. The child screamed violently when he attempted to make a physical examination, and it was with difficulty that he could make out the existence of broncho-pneumonia. The liver appeared to be enlarged and the spleen as well. No fluctuation could be elicited in the liver. The temperature was 105°, pulse very rapid and weak; great dyspnœa; face pale; lips and gums quite bloodless.

After death the following conditions were found: The liver, before opening the abscess, weighed two pounds, ten ounces. The abscess was situated in the right lobe, protruding but slightly above the upper surface of the liver, and extending, posteriorly, to the border of the lobe, and, anteriorly, to within about two and one-half inches of the edge. The cavity contained seven ounces of laudable pus. Connected with the main abscess, by a narrow channel, was a small abscess containing about half an ounce of pus. This small abscess existed anteriorly to the larger one, thus bringing it to within one and one-half inches of the anterior border of the right lobe of the liver. It did not project beyond the surface of the liver. Spleen weighed five ounces two drachms; was firm in consistence; showed no reaction to the iodine stain for amyloid degeneration. Kidneys showed nothing abnormal. The lung, which was examined microscopically, proved to be affected with catarrhal pneumonia. No evidence of inflammation anywhere above the diaphragm that might have extended through to the liver.

The points of special interest to which I would call attention are as follows: The abscess occurred in a young child. It occurred in a female. It was the result of external violence.

December 9, 1886.

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20. *Abscess of the liver.*

Exhibited by Dr. W. E. Hughes.

The patient had been seen by Dr. M. B. Musser, a short time previous to his death, and to him I am indebted for the clinical history and the specimen. The patient, a man, æt. 45 years, had an attack of what was at the time, supposed to be an irregular typhoid fever. He recovered from this, but was shortly afterward taken with fever, and rapidly

maciated. A few days before his death a swelling developed in the epigastrium, and, abscess of the liver being suspected, this swelling was cut down upon, but no pus found. The next day the incision in the abdominal wall opened and there was a free discharge of pus.

At the autopsy the whole left lobe of the liver was found completely hollowed out by an abscess. This abscess had ruptured, but free escape of pus into the abdominal cavity had been prevented by firm adhesions between the abdominal wall, intestines, and liver.

December 9, 1886.

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21. *Three cases of abscess of liver.*

Exhibited by Dr. W. Osler.

Case I. *Chronic dysentery; small abscesses of left lobe; two of them communicating with duodenum.*—C. B., æt. 41 years, baker, was admitted to the Philadelphia Hospital August 9, 1886. He had typhoid fever when fifteen years of age. Two years ago the present trouble began with pain in abdomen, vomiting, and diarrhœa. At times he would be better, but not for long. There was blood in the stools at first, and he had much burning pain in the rectum during defecation. At time of admission he had four or five soft stools daily, not containing blood. He had slight fever at first, but subsequently the temperature was normal. He had lost flesh, and had a sallow, cachectic appearance. He had chills and fever shortly after the trouble began, but none during his stay in the hospital. On September 18th, the following note was made: Abdomen flat, veins not distended; epigastrium prominent, and on palpation a firm, smooth mass is felt, occupying the entire region from the navel to the sternum. An indistinct edge can be felt a little to the right of, and also below, the navel. To the right the mass does not reach the nipple line, and here the fingers can be placed beneath the costal border. The mass is extremely tender; it moves with inspiration. In the sternal and right parasternal lines the liver dulness is directly continuous with that of the mass in the epigastrium. In the nipple and mid-axillary lines there are four and five inches of liver dulness. On October 11th, the note was: Mass above the navel rather more prominent, still very tender; it cannot be separated from the left lobe of liver; right lobe certainly not enlarged. The vomiting was variable and latterly was not so troublesome. He continued to have two or three soft

motions daily. The emaciation and weakness became most profound, and he died on November 10th. Post-mortem notes as follows:

Body extremely emaciated; abdomen depressed. On incising the abdominal wall, an abscess was opened, which is between the peritoneum and right lobe of liver, and extends for an inch from ensiform cartilage to navel; this communicates directly with an abscess cavity in the right lobe of the liver. There is no peritonitis. Omentum is adherent at the right lobe. The right lobe of the liver does not extend beyond the costal border. The left lobe extends three inches below the ensiform cartilage.

Stomach: A little distended; contains a quantity of semi-digested food. The pylorus is free, mucous membrane soft and congested. In the œsophagus just above the cardiac orifice, there is an oval tumor, about the size of a bean, which is submucous, freely movable, and on section is firm, grayish-white in color; looks like a sarcoma. The duodenum is normal. Orifice of the bile-duct pervious; a clear bile flows out. The duodenum, one inch below pylorus, is closely adherent to the hilus of the liver, and on squeezing this organ pus flows from two orifices through which a led pencil could be passed. The tissues in gastro-hepatic omentum thickened. The hepatic artery not increased in size. The portal vein is pervious; both branches free. No thrombi.

Spleen: Is enlarged, is amyloid in reaction.

Liver: Not enlarged. Tissues about suspensory ligament thickened. The anterior portion of left lobe is adherent to the abdominal wall, and abscess cavity has perforated from the liver substance and is directly in contact with the abdominal muscles. It was this mass that was evident externally. This abscess is the size of an orange and contains a creamy pus; it is lined by a thick membrane; it communicates directly with the duodenum; it is confined to the lower and anterior part of the lobe. The right lobe of the liver is firm and is amyloid in reaction.

The *colon* is thickened, the mucosa roughened, irregular, and in many places ulcerated—evidently an old dysenteric condition.

The prominence of the gastric symptoms, the distinct tumor, and the progressive emaciation, led to a diagnosis of cancer of the stomach in this case. On two occasions a careful examination was made with a view of deciding whether the mass was associated with the liver, but it was not found practicable. Sufficient stress was not placed upon the intestinal symptoms, which had, however, passed the acute stage. The extreme sensitiveness of the mass is a point worthy of note, as it was much more than the ordinary pain and tenderness of malignant disease.

Case II. *Acute dysentery; three recent abscesses in liver.* — Hugh McB., æt. 50 years. Admitted November 16, 1886. Had been ill about ten days with diarrhœa and great pain in the abdomen. He had kept about and tried to work until a few days before admission. Had always been healthy, but had been a very heavy drinker. When seen on the 17th, appeared to be in collapse, face covered with sweat, tongue furred and dry; respiration 50; pulse 130, very small. Abdomen greatly distended, tense, and drum-like, and extremely tender. On percussion, tympany everywhere; liver pushed up; area of dulness diminished. There had been vomiting, which was now checked. The diarrhœa still continued; the stools small; much mucus and some blood. The temperature was 102° on the evening of admission, sank to 98° on the morning of the 17th, and rose to 100° on the evening just before his death.

Body that of a middle-aged, well-nourished man. Abdomen distended. Right inguinal hernia. Coils of intestines greatly distended, and here and there flakes of lymph over surface. Large intestine enormously distended, the transverse portion equal in size to thickest part of arm. In spots the peritoneal surface was covered with lymph, and the wall of gut had, in places, a grayish sphacelated look. The small intestine removed; no adhesions; it was dilated, swollen.

Large intestine: Cæcum was thickened and inflamed. The membrane was in enormous folds transversely placed, blackish in color outside, and sphacelated in places to a depth of from three to five lines. This condition extended up the ascending colon and was very marked, also, in transverse and descending portions. Some of these necrotic areas were isolated and ranged from a quarter to half an inch in size, and presented a cap of dark gangrenous tissue surrounded by a zone of grayish-white tissue from a quarter to half an inch in diameter. The necrosis in some of these spots extended through to the peritoneal coat.

On right side a knuckle of the lower portion of the sigmoid flexure was nipped in the inguinal ring, projecting about an inch beyond the level of Poupart's ligament. The hernial sac, when opened, was about the size of a small orange, contained lymph and serous fluid, and at its upper part the knuckle of intestine thickly covered with lymph. The orifice of the ring was large; the portion of gut was adherent only to the inner and lower aspect.

On slitting open the sigmoid flexure and rectum, a knuckle of bowel was adherent to ring as above described. From within, the mucous membrane was dark and gangrenous; the gut for a distance of three or four inches above and below this part was greatly thickened; the mucous membrane sphacelated.

Stomach and duodenum presented no special changes. The small intestine was swollen and presented no lesion of the mucous membrane.

Liver looked normal. In the right lobe were two spots, the size of large marbles, of a grayish-brown color, well defined from liver substance, yet without a limiting capsule. These spots represented the commencement of a hepatic abscess. On further incision of organ there were three recent abscesses, the size of oranges, softening at centre and presenting fragile necrotic liver tissue at periphery.

Case III. *Chronic phthisis; multiple small abscesses in liver.*—M. B., admitted to Philadelphia Hospital, June 6, 1885. The clinical history of the case is of interest from the early and persistent symptoms of dropsy associated with disease of the kidneys. The autopsy showed extensive disease in both lungs and large amyloid kidneys. The liver was a little enlarged and on section presented very many small abscesses, none larger than a marble, containing a creamy, in places, bile-stained pus. They could not be traced in connection with the portal vein, the branches of which were free. There were a few ulcers in the lower part of the ileum. The mesenteric veins were not involved; no inflammation about appendix or pelvis. *December* 9, 1886.

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22. *Two cases of cancer of the stomach.*

Exhibited by Dr. W. Osler.

Case I. *Large open cancer of anterior wall and lesser curvature.*—P. H., æt. 52 years; laborer. Admitted to the University Hospital in April, 1886. Family and personal history excellent. For three months had pain in abdomen with frequent eructations. Appetite poor; gradual loss of flesh and strength. When admitted was fairly well nourished and muscular; face pale. Inspection showed a depressed abdomen, with marked fulness in umbilical region, which was altered somewhat in shape, with a peristaltic movement. Palpation revealed a firm, hard tumor in epigastric region extending to the navel; uniform, not nodular. The centre of the mass was about an inch and a half above the navel, and it had a percussion limit three inches in diameter. There was moderate dilatation of the stomach. The liver, spleen, and the thoracic organs were normal. He remained in the hospital nearly four months. At first the vomiting was very troublesome, but toward the end it became much less frequent. The vomited matter contained an extraordinary number of

the sarcina ventriculi. During the last month of his life diarrhœa was severe. He emaciated rapidly and died early in August.

The stomach was large and the specimen showed a large ulcerating cancer of the anterior wall and lesser curve, extending to the pylorus. There were several soft sloughs, and it was only at the edges of the mass that the infiltrated thickened wall had a cancerous appearance. There were no secondary masses in the organs.

Case II. *Annular cancer of pylorus.*—A. B., æt. about 56 years, patient of Dr. Bolling, was seen in May, 1886. He had been ill for nearly a year with dyspeptic symptoms and failing health, and there had been considerable difference of opinion as to the nature of his illness. At the first visit the most striking feature was the profound anæmia, with very moderate wasting. There was pain after eating and occasionally vomiting, but with careful dieting the latter symptoms were readily controlled.

Examination revealed a small, hard, tender nodule, about two inches below the ensiform cartilage. The stomach did not appear to be dilated. The general and local symptoms were thought sufficient to indicate organic disease of the stomach. I saw him on three occasions and on the last the small mass in the epigastric region was still more distinct, and the condition had improved somewhat. I did not hear anything more of the case for four months, when Dr. Bolling asked me to be present at the autopsy. The stomach symptoms had become more pronounced and the emaciation extreme. The disease was found limited to the stomach, in the pyloric region. The sections here shown illustrate its characters. The pylorus was involved with the duodenum for an inch, and the stomach for at least two inches, forming a uniform annular mass. The little finger could be passed into the pylorus, but, as the sections show, the lumen was much narrowed by the projection of irregular masses, which gave a sinuous outline to the cross sections.

December 9, 1886.

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23. *A case of gall-stones with extreme dilatation of the biliary canals.*

Exhibited by Dr. J. H. Musser.

Dilatation of the bile-ducts by gall-stones; ducts patulous; gall-bladder atrophied; no symptoms of hepatic colic; death from acute disease.

The patient, a female, æt. 73 years, was admitted to my wards in the

Philadelphia Hospital, with acute pleurisy with effusion. She had been an inmate in another department of the hospital upward of ten years. Her associates do not recall that M. L. had ever had biliary colic or jaundice. The acute disease caused the death of the patient, and at the autopsy the following appearances of the abdominal organs were observed:

Stomach somewhat contracted; contained curdled milk; mucous membrane normal.

Duodenum: slight congestion of mucous membrane.

Papillæ enlarged and prominent; no appearance of cicatrix or recent ulcer; bile oozed on pressure of ducts.

Liver not enlarged; surface smooth; contour normal; color fair; edges discolored by decomposition. At one point adhesion of capsule to the diaphragm; otherwise capsule normal.

Common duct, dilated to the size of the gall-bladder, contained two large gall-stones the size of a small walnut; each stone filled a sacculation. Bile could be gently forced past the stones. The hepatic duct before subdividing readily admitted two fingers. In its dilated lumen one large and eight or ten small stones were lying. The cystic duct was widened and apparently shortened, leading to the small gall-bladder, which was in appearance almost like a continuation of the cystic duct—a diverticulum of the main duct. In the contracted gall-bladder a gall-stone was firmly fixed and surrounded by muco-pus. The fundus of the gall-bladder was adherent to the colon. The mucous membrane of the common and hepatic duct was changed in character—roughened and ribbed, not unlike the appearance of the urinary bladder in old age. The branches of the hepatic ducts were dilated to their terminations in the periphery of the liver, and filled with gall-stones, which were not of recent formation. They did not, however, end in club-shaped dilatations or sacculations, nor did the surface of the liver present indications of such condition of the ducts. The larger branches of the hepatic duct readily admitted a finger. The dilatation and collections of gall-stones were so uniform, that wherever section of the organ was made, stones were cut upon. Quite a collection of stones existed in the only duct that approached a cystic character underneath the previously mentioned localized perihepatitis. The liver structure was not much changed. The acini were compressed and the veins evidently pressed. There was no evidence of cirrhosis, but with a lens an increase of the connective tissue about the ducts was apparent. Portions were reserved for microscopical study, a report of which will be made in the future.

January 13, 1887.

24. *Gall-stones; a number of specimens, with some clinical histories.*

Exhibited by Dr. J. H. Musser.

The collection of stones which are presented, it was thought, would be of interest, as they illustrate the various chemical and physical characters of gall-stones, and also some of the pathological results of their presence in the body.

Your attention is first called to the gall-bladder here exhibited. The cystic duct was contracted in the duodenal two-thirds, and a mucopurulent fluid exuded on pressure. The inner third was dilated, and included firmly in its lumen the large gall-stone in box No. 1. Back of the impacted stone the neck of the bladder was thickened and contracted, and formed a barrier between the impacted stone and the gall-bladder, which contained a serous fluid and eight stones with facets. The normal mucous membrane was converted into a smooth, shining, serous-like membrane, which secreted serous fluid. The patient from whom these stones were removed was a female, æt. 42 years, who died of pneumonia. There was no history of previous hepatic disorder. The large impacted stone bore no facets.

No. 2. Stones removed from a man who died of an acute disease. He was past middle age, and had no knowledge of biliary colic. They are compound stones, five in number, each the size of a hazelnut.

No. 3. During life the patient presented symptoms supposed to be due to gastric carcinoma. A female, æt. 52 years, of good habits and comfortable circumstances, married, was seized one year before her death with pain in the epigastrium, and vomiting. The former was paroxysmal and lancinating, and continued throughout the year. The latter was also persistent, and the matter ejected was not the food, but an acid fluid. The vomiting occurred one hour or more after eating. Blood was never vomited. While under observation a hard, fixed, somewhat irregular tumor was detected in the epigastrium. As the patient wasted the mass became more prominent. It was tender on pressure, but did not yield thrill or murmur. It was quite superficial, and about the size of a small fist. During the progress of these symptoms the patient became extremely emaciated and weak, and finally died of exhaustion. Clinical diagnosis: cancer of the stomach. Neither biliary colic nor jaundice was present during the patient's life.

The attending physician asked me to make the autopsy, which was limited to the abdominal cavity. The body was pretty well decomposed.

The tumor observed during life was in the median line, and adherent to the left lobe of the liver and the stomach. It was found to be an enlarged gall-bladder with thickened walls. It had been twisted directly around, the fundus pointing to the spleen. Considerable local peritonitis of old date (?) was present. The ducts of the liver were pervious, the cystic duct contracted. The gall-bladder was distended by sixty gall-stones, and some mucoid fluid. The stones present facets; they are compound, and are irregular in shape.

No. 4. The patient from whom these stones were removed after death was about 50 years of age, and died of strangulated hernia. The cystic duct was impervious. The gall-bladder firmly surrounded the gall-stones, and was quite contracted on them. The stones were made up almost entirely of cholesterin.

No. 5. Two stones removed after death from a man, æt. 70 years, who never had biliary colic. A uric acid calculus was also present in the urinary bladder. This combination has been observed before. The gall-stones were compound and laminated.

No. 6. Large, single oval-shaped gall-stone, with rough surface. Patient, a female, æt. 55 years, died of carcinoma of the pylorus. There had never been any jaundice. The bladder contained only the stone, and was firmly contracted around it. Cystic duct was pervious, but not bile-stained.

No. 7. Thirty laminated compound stones removed from a patient who died of acute disease, and was never aware of the presence of the calculi in the gall-bladder. Female, æt. 45 years. Gall-bladder and ducts normal.

No. 8. Forty-two gall-stones composed chiefly of bilirubin, a few with alternating layers of cholesterin. Removed from a blind woman, æt. 60 years, who had a history of previous hepatic colic and of local peritonitis. The latter was seen after death, and these stones observed, although there was no stricture of the ducts. Patient died of organic brain disease.

No. 9. Granules of bile-pigment. The gall-bladder was filled with these granules. Harley dwells on the clinical features observed in the passage of this pigment. *January* 13, 1887.

25. *Two unique gall-stones (white).*

Exhibited by Dr. W. A. Edwards.

The biliary calculi which I exhibit this evening were passed by a patient under the care of Dr. Walter F. Atlee. The man, æt. 30 years, had frequent attacks of hepatic disorder; during one of these, about three weeks ago, he passed these two perfectly white, glistening, shining gall-stones, three-quarters of an inch in diameter.

White gall-stones are somewhat rare; the *Transactions* of this Society for fifteen years (1871 to 1886) do not record a single specimen of these colorless stones.

J. Wickham Legg records a white gall-stone, and Ford T. Roberts seems to have had unusual good fortune in this respect, in so much that he concludes that, as a rule, gall-stones have a brownish or greenish-yellow color, and are opaque; but they present an endless variety of tints, ranging from white to black, blue, green, red, and other colors. Occasionally they are somewhat translucent. These stones seem to be made up of carbonate of lime and cholesterin; the latter may be nicely studied in the specimens after having been acted upon by sulphuric ether or boiling alcohol. *January* 13, 1887.

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26. *Fibroid thickening of the pylorus.*

Exhibited by Dr. J. C. Wilson.

The patient, a white male, æt. 66 years, had been in good health till within eighteen months of his death. Then he began to complain of dyspeptic symptoms, fulness in the epigastrium, weight and discomfort after eating. After awhile vomiting came on, never during the day, but at night after he had assumed the recumbent posture, when large quantities of a yeasty fluid would be ejected. The vomited matters contained sarcinæ, but never blood. There was at no time pain. Examination of the abdomen showed a tumor, as large as a pigeon's egg, in the epigastric region usually situated just to the left of the median line near the margin of the ribs. It was freely movable, and when the stomach was fully distended with food would be found in the median line, or to the left, just above the umbilicus. The patient became extremely emaciated, and had to be fed almost exclusively by the rectum. Surgical interference

was urged, but owing to objections by relatives, was abandoned. Death resulted from inanition.

At the autopsy the tumor was found to be an enormous development of fibrous tissue about the pylorus. The pyloric orifice was much narrowed, barely permitting the passage of a small pencil. The stomach was enormously dilated. *January* 13, 1887.

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27. *Perforation of vermiform appendix: retro-cæcal abscess.*

Exhibited by Dr. W. E. Hughes.

The patient from whom this specimen was obtained was seen by Dr. J. H. Musser, a few days before death. He was a man, æt. 20 years, who had been employed in an ice house. For more than a week before his death he had felt indisposed, complaining of fever, loss of appetite, distention of abdomen, and colicky pains. Thinking himself "bilious" he had purged himself vigorously, but without feeling any relief. When first seen his abdomen was distended but there was no marked tenderness. The hypogastric region was dull on percussion, and in the right iliac fossa there was a sense of indistinct resistance to pressure.

At the autopsy, the intestinal coils were firmly matted together by recent adhesion. The inflammation was most marked in the pelvis, where there was quite a quantity of purulent exudation. About the cæcum the intestines were very firmly glued together, and behind the cæcum in the cellular tissue, was an abscess cavity as large as an English walnut, filled with pus having a distinctly fecal odor. The abscess cavity did not communicate with the peritoneal cavity. Along the inner wall of the abscess ran the vermiform appendix, its tip curving round and lying free in the cavity. At one-quarter inch from its free extremity it had perforated, the perforation communicating with the abscess. The solitary and agminated glands in the lower part of the ileum were congested and swollen, one of the patches near the ileo-cæcal valve just commencing to ulcerate. The appearances in the intestine were identical with those of the early stage of typhoid fever. The large intestine was healthy. The other organs were not remarkable.

January 13, 1887.

28. *Two cases of abscess of the liver.*

Exhibited by Dr. H. F. Formad.

Case I.—Of traumatic origin. The patient, a male, fell from a cart striking his side heavily on the wheel. He felt a little uneasiness in his side but kept at his work for a month. Then he became worse and was confined to his bed for four months before his death. During this time the abscess was tapped repeatedly and gallons of pus removed. He died of rupture of the abscess into the peritoneal cavity and consequent peritonitis.

At the autopsy two abscess cavities were found in the right lobe of the liver, each as large as the largest orange. They were filled with pus, in which floated masses of liver cells.

Case II.—Following dysentery. The patient had an attack of acute dysentery, which was followed by a period of apparent cure, then by fever, hectic, and emaciation. Death resulted from rupture of the abscess with peritonitis.

At the autopsy a single abscess cavity was found in the left lobe of the liver. In the colon were found the lesions of chronic dysentery—thickening and ulceration throughout its whole extent.

January 13, 1887.

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29. *A case of poisoning by hydrocyanic acid.*

Exhibited by Dr. H. F. Formad.

It was unknown how long the ingestion of the poison had preceded death. The body exhaled a perceptible odor of the poison, which was much increased on incising the tissues. Throughout the œsophagus, stomach, duodenum, and upper part of the jejunum was a very severe and universal hemorrhagic infiltration of the mucous membrane. This reached its maximum in the stomach, and in the jejunum, two feet from the pylorus, gradually faded off into normal mucous membrane. In the pancreas was seen this same hemorrhagic infiltration, with here and there between the lobules points of actual hemorrhage. The kidneys and spleen also showed hemorrhagic points. The heart was dilated and distended with liquid blood. All the blood throughout the body was absolutely liquid and had a strong odor of the acid. The organs, gener-

ally, were deeply stained with the blood-coloring matter. The lungs were deeply congested.

The most remarkable points in these cases of hydrocyanic acid poisoning, are the direct local effect of the acid upon the mucous membrane, and its secondary effect upon the blood. The action upon the mucous membrane begins in the fauces and reaches as far as the poison has gone, culminating in the stomach. The effect is very similar to that of arsenic, but less severe. The blood corpuscles are partially disintegrated, and the pigment to some extent freed, accounting for the extensive staining of the tissues. This staining power I have taken advantage of, and all the red ink I use in my laboratory is the blood of victims of hydrocyanic acid poisoning. The preservative power of the poison on the blood is very remarkable—a specimen two years old is still perfectly fresh and does good duty as red ink. *January* 27, 1887.

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30. *Impacted biliary calculi; suppuration in gall-bladder and bile-ducts; death.*

Exhibited by Dr. J. Tyson.

The stone exhibited, which is almost spherical, and half an inch in diameter, was one of two removed post-mortem, one from the junction of the cystic and hepatic duct, the other from the common bile-duct about an inch further down, from a lady, æt. 55 years, under the care of Dr. W. V. Woods, of Odessa, and Dr. Valandingham, of St. George's, Delaware. Dr. Tyson first saw the case with these gentlemen on the morning of January 16th.

When seen she was moribund and intensely jaundiced. There was exquisite tenderness over the region of the liver, and especially at its lower portion. There was no enlargement of the liver, but it was still thought there might be cancer of the liver, or cancer of the head of the pancreas. This proved not to be the case.

The patient died on the afternoon of the same day, and at the autopsy made by Dr. W. V. Woods, the following was noted: The duodenum was congested, as was also the pyloric end of the stomach; the peritoneum in the vicinity was inflamed, and had formed adhesions which were easily broken up. On looking for the gall-bladder, the common bile-duct was first seen, distended with these stones, and with pus, the stones being situated as described. The gall-bladder was also distended with pus, having at least an ounce or more in it. *January* 27, 1887.

31. *Cirrhosis of the liver.*

Exhibited by Dr. J. H. Musser.

A woman, addicted to the inordinate and habitual use of alcohol. There had been a period of dyspepsia and failure of health, followed by considerable peritoneal dropsy. There was no jaundice

At the autopsy the liver was found small, hard, and with the characteristic nodular appearance of its surface. On section, considerable amounts of hepatic tissue were seen to be replaced by fibrous tissue. The portal vein was much narrowed. There was extensive perihepatitis and perisplenitis, but no enlargement of the spleen.

January 27, 1887.

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32. *A case of obstruction of the bowel from adhesions of the ileum to the right ovary; death ten days after delivery.*

Exhibited by Dr. D. Longaker.

Rebecca B., æt. 34 years, entered Philadelphia Lying-in Charity in her second pregnancy, a few days before her anticipated confinement. She made no complaint of any kind during this time, and assisted in the light duties of the house. She was rather poorly nourished.

January 15, 1887. The patient was in pain; examination showed the os patulous and the head presenting. She vomited her supper. On the following day, feeling bad, she remained in bed, vomited several times, and by midnight was actively in labor. The usual enema was without effect. At the close, or near the close, of the first stage of labor, the nausea was excessive, and there was occasional bilious vomiting.

17*th.* At 6.30 A.M., after a short expulsive stage, an eight-pound male child was born. Nausea continued during the day, and she was unable to retain food.

On the second day she was placed on lime water and milk. She complained of slight pains in the lower portion of the abdomen.

On the third day an enema was given, with very slight effect.

Fourth day. Catheterization, which had been necessary during the first three days, was no longer required. As the lochia had acquired an offensive odor, a 1 : 4000 bichloride of mercury injection was given every four hours.

During the next three days she was quite comfortable, free from pain, and took liquid food without emesis. There had been no stool for nine days. There was tympany in the umbilical region. As enemata and the post-partum pill of Fordyce Barker had been used without effect, a large enema, with the patient in the knee chest position, was ordered. Scarcely had the patient assumed the recumbent position when a considerable quantity of yellow fluid gushed simultaneously from the mouth and the anus. Its odor was very offensive.

Her temperature was subnomal in the evening, 96.8° F. It had been normal since delivery. Stimulants and external warmth were administered during the night. On the following morning at 8 o'clock, temperature 95.8° F.; at 9.30 A.M., 96.2° F.; at 12.40 P.M., 97.6° F.; at 5 P.M., 97.8°. A severe attack of vomiting came on at 5.10 P.M.; she had been entirely free from this symptom since the preceding day. She was freely stimulated, and external warmth was continued. At 10 P.M. temperature 98° F., no pain. Tympanitic distention, as before noted, slight, and limited to the umbilical region.

The advisability of laparotomy was discussed, but it was decided adversely. This was the ninth day after delivery. A specimen of urine withdrawn by catheter proved albuminous. During the night there was slight hemorrhage from the vagina.

Tenth day, 7.15 A.M. Pulse 150; temp. 101° F. At 3.30 P.M., vomiting again, followed by severe pain. She died of exhaustion at 9.25 P.M.

Post-mortem examination, forty hours after death.—The abdomen was slightly distended. There was considerable subcutaneous fat. There was a recent slight peritonitis.

The *stomach*, *duodenum*, *jejunum*, and *ileum*, with the exception of the lower portion of the latter, were enormously distended. At several points in the distended ileum minute perforations were observed, from which oozed the semifluid contents on pressure. These were evidently post-mortem. None of the contents had escaped prior to the handling of the intestines. Three feet above the ileo-cæcal valve an obstruction was found. This was produced by two bands of adhesion, attached four inches apart from each other, and passing from the gut to the right ovary. The loop of bowel was behind the uterus, in the pelvis. It was in a gangrenous condition. The intestinal canal beyond this point was very much contracted. The ascending colon fitted my little finger like a glove.

Uterus and appendages: The fundus was on a level with the brim of the pelvis, the normal degree of involution for the tenth day. The tube and ovary of the left side were normal. The right tube was slightly

swollen, and the ovary on this side was the size of a hen's egg, and cystic. In it was found a recent blood clot about ten days old. The appendages on both sides were bound to the posterior surface of the uterus by moderately firm adhesions.

The *endometrium* was in a healthy state. The spleen, liver, and kidneys were normal.

Remarks.—The adhesions of the ileum to the pelvic viscera had existed, certainly, for some months. The changed relation of the parts after delivery rendered the obstruction complete. What is especially noteworthy in the history of the case, is the absence of symptoms of strangulation. Had these existed, there would hardly have been a question of the propriety of laparotomy. *February* 10, 1887.

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33. *Hernia of cæcum and appendix; perforation of latter; old perityphlitic abscess; recent larger one; general peritonitis.*

Exhibited by Dr. W. Osler.

C. J., æt. 50 years; cook. Admitted to University Hospital, March 4, 1887.

On Sunday night, February 27th, after having taken a walk, was suddenly seized with intense pain in hypogastric region. Immediately took to his bed, vomited two or three times, and on the following day noticed that his abdomen began to swell. On Wednesday night had forty stools. Dr. Wharton gave him a hypodermic of morphine, which relieved the pain for that night. On Friday was admitted to hospital. Abdomen greatly distended and tympanitic. Breathing rapid and difficult. Pulse 112. Complained of great pain, chiefly in hypogastric region. Temperature 100°. Was given enemas of turpentine with sweet oil; whiskey every two hours; poultice over abdomen.

March 5. Dr Wharton gave history of the existence of right inguinal hernia. Temp. to-day, 99°. Feels much relieved, but still complains of pain and difficulty in breathing. Vomited this morning twice; dark-green in color, but no fecal odor. Bowels have not been opened.

6th. Complains this morning of intense pain. Is vomiting constantly. Was given a half grain of morphine in two hours, which relieved him for a short time. Hoffmann's anodyne and rectal tube gave no result in relieving tympanites. Punctured three times without relief. Died that night at 11 P. M.

Autopsy.—Well-built man; abdomen distended. In nipple line not more than two finger's-breadth of liver dulness. Three punctures of abdominal wall, one in right lumbar region and two in epigastric. On opening peritoneum gas escaped; moderate amount of fluid, chiefly in flakes, sero-purulent. Coils of small intestine greatly distended, one crossing at the level of navel as large as arm above wrist; toward pelvis not so large. The general surface of intestines was injected. Peritoneum coated in places with recent lymph. Colon moderately distended. Cæcum and ascending colon contained consistent feces, those in former very dry. Cæcum was adherent to the iliac fascia and passed into ring, and was adherent in it for the distance of two inches. Ileum looked normal, and finger passed freely through ileo-cæcal valve.

The *liver* was separated from parietes by an air space. Over promontory of sacrum the coils of intestines were closely adherent, and between and behind them there was an abscess cavity, circumscribed, and walls partly formed by folds of mesentery. Abscess extended into Douglas's pouch. Bladder contracted, walls thickened. Sigmoid flexure and rectum opened; no perforation; walls of abscess were formed by several inches of sigmoid flexure; ureters not involved. In searching for appendix the proximal orifice was found at the extreme end of the hernia of cæcum, in the inguinal canal; it then curved upon itself, passed back into abdomen immediately behind terminal portion of the ileum; passing to the left it was adherent directly to abscess cavity. The lumen was free; terminal three-quarters of an inch thick, sloughing, and opened directly into a smaller circumscribed sac with pigmented and indurated walls, the size of a large walnut, which communicated directly with larger one. The boundaries of the larger cavity were formed by mesentery and ileum in front, sigmoid flexure and retro-peritoneal tissues behind. It was along the sigmoid flexure that the abscess passed into Douglas's pouch.

Spleen large and soft.

Kidneys somewhat swollen.

Heart valves normal.

Lungs normal.

Remarks.—We have here an illustration of a very common event in perityphlitis, viz., the occurrence of general peritonitis by extension of inflammation from a localized focus of suppuration, the result of perforation of the appendix. The smaller circumscribed abscess behind the terminal part of ileum, into which the appendix opened, had doubtless existed for some time, and may not, when forming, have excited serious symptoms. The frequency with which we find evidence of past appendicitis, indicated by adhesions, fibroid and pigmentary changes, and ob-

literation of the distal part of the tube, points to recovery as the termination of a considerable proportion of the cases of perforation.

The hernia of the cæcum and appendix is an interesting point, and the curious course taken by the latter made the dissection very difficult, and it took both time and care to make out the precise relationship of the parts.

The fact that he had an old hernia, which Dr. Wharton had at one time reduced, suggested the possibility of the trouble originating about it. Laparotomy was advised, and Dr. Ashhurst came out one evening for the purpose, but the patient refused his consent.

February 24, 1887.

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34. *Primary melanotic sarcoma of the eye; extensive secondary deposits (melanotic) in the liver; smaller foci in the skin, pancreas, spleen, kidneys, and heart.*

Exhibited by Dr. J. Tyson.

The specimens were derived from a man, æt. 73 years, who was admitted to the medical wards of the Philadelphia Hospital November 7, 1884, and died March 3, 1887. He was jaundiced, and the pigmentation deepened until he died. The urine was intensely jaundiced, and the liver was enormously enlarged. The right eye was evidently diseased, but, as the man was a prize-fighter, and gave the history of a blow received some time ago, it was not closely examined.

Autopsy (by Professor Osler) revealed the following: Body that of a medium-sized elderly man. Skin deeply jaundiced. Legs, thigh, and back œdematous. Over skin of the thorax five or six pigmented bodies, varying in size from a pin-head to a large pea.

Abdomen: Intestine distended, small amount of bloody exudation. Liver projected five or six inches below costal margin; left lobe in nipple line at the heart four inches below costal margin; surface of liver uniformly dark in color. Diaphragm corresponded on left side to fifth rib, and on right to the fourth.

Thorax: In the right pleural cavity an effusion of bloody fluid; on left side a few ounces of same.

Pericardium: Beneath visceral layer a number of small dark bodies, varying in size.

Heart: Chambers contained fluid blood and clots. Valves on right side normal. Endocardium bile-stained. Edges of tricuspid valve

thickened. Beneath endocardium of the right ventricle numerous small miliary bodies, some pale, most of them dark in color. On the left side mitral valves were thickened, the aortic a little opaque. Beneath endocardium numerous large secondary masses. Throughout substance many nodular growths, none larger than peas.

Lungs crepitant, œdematous, congested posteriorly; no secondary nodules. *Right* collapsed; flakes of lymph covered surface. No cancerous nodules. A few pigmented nodules on costal pleura.

Stomach distended. Mucous membrane soft. A few pigmented spots scattered throughout the mucous membrane.

Duodenum contained much mucus. The papillæ were swollen. Pressure on the bile-duct caused a clear fluid to flow. No obstruction. Gall-bladder almost empty; numerous pigmented masses throughout it. Hepatic artery large. Portal vein free, admitted little finger readily.

Pancreas presented numerous secondary nodules, some as large as beans.

Spleen mostly adherent to diaphragm. Secondary mass as large as small walnut in one end.

Kidneys: In *left*, many small pigmented bodies in fatty capsule; organ tough; arteries thick; a few cysts through substance; here and there a few pigmented bodies. In *right* kidney there were secondary nodules, some as large as marbles.

Retroperitoneal glands were not enlarged, and not melanotic.

Aorta: Numerous flakes of atheroma.

Right suprarenal capsule removed with the liver. Both of the capsules were normal.

Liver: Shape of organ retained. Right lobe larger in proportion than the left; surface smooth. Capsule thickened in places. Numerous small superficial indentations. Color was uniformly dark, here and there having yellow spots. There were some button-like projections which were surrounded by distinct depressions. These prominences were extremely hard. Section through substance showed it to be tough. The surface showed: 1st. Large irregular deeply pigmented areas, which occupied the greater portion of the surface. Some of these were intensely black. Other areas were mottled with grayish-white. 2d. Irregular areas of liver substance, greenish-yellow with black specks scattered through them. In upper and back portions of right lobe and in left lobe were considerable areas of liver substance. Organ weighed seventeen and three-quarters pounds.

Brain: In right frontal area of skull-cap, two pigmented areas in the diploë.

Dura: Several small pigmented masses adhering to under surface of dura, one of these, the size of a pea, compressed the angular convolution. At the base of skull, in anterior and middle fossæ, were from ten to twelve small pigmented bodies. In the left fossa small but well-marked hemorrhagic pachymeningitis. Arteries at base of brain atheromatous. Section of brain, no secondary masses in substance.

Eye, right: Lower part of conjunctiva showed a pigmented mass. This passed backward, and reached as far as entrance of the optic nerve. It was lobulated, and looked as if it had perforated the sclerotic. Old cataract with anterior synechia and obliteration of pupil. On section of eye the whole ball behind the lens was occupied by a pigmented mass, which was intensely black except at one spot. It had perforated the sclerotic at one point, and grown out beneath the conjunctiva.

March 10, 1887.

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35. *Abscess of the liver.*

Exhibited by Dr. E. T. Bruen.

The specimens herewith presented were taken from the body of a man æt. 56 years, who was admitted to the Philadelphia Hospital April 4th. During the months of October and November he had been treated for dysentery in the hospital, under Dr. Osler. The treatment was by rectal injections, and resulted in entire relief, so that the patient was discharged from the hospital. When admitted, the temperature was found to be 104°, there was some delirium, and during the three days succeeding his admission the temperature never fell below 102°. There were no chills or sweats during this period. He had, however, a dysenteric diarrhœa, the passages being very frequent. The liver was found to be enlarged, especially in an upward direction, and in the axillary and infrascapular regions.

Abscess of the liver being suspected, the needle of an aspirator was inserted in the eighth interspace, and about sixteen ounces of chocolate-colored fluid evacuated, after which, a drainage tube was inserted. There was no relief from the unfavorable symptoms of fever and diarrhœa, and the patient died forty-eight hours after the operation.

At the autopsy a large abscess cavity was found in the upper surface of the right lobe of the liver. The diaphragm was closely adherent to the liver, but the pleural surfaces were normal. This abscess had been entered by the needle of the aspirator, which had traversed the lower

part of the pleural cavity without damage. In the left lobe of the liver another abscess cavity was found, about as large as an orange. This latter cavity was round and circumscribed, the former was large and irregular. The large intestine was also examined, and found covered with healed ulcers, which testified to the thorough action of the nitrate of silver injections. The other organs were normal.

March 24, 1887.

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36. *Intestinal ulcers.*

Exhibited by Dr. F. A. Packard.

Specimens removed from the body of Jane McK., æt. 34 years, dead of phthisis pulmonalis, at Pennsylvania Hospital. Patient was under observation from November 31, 1886, until her death, May 10, 1887, and had never had any tendency toward diarrhœa. Among other lesions there were found at the autopsy, numerous crater-like ulcers of the size of a split pea, and extending down to the subperitoneal coat of the small intestine. Peyer's glands presented typical "shaven-beard appearance." In caput coli there were two large shallow blood-stained ulcers, while in the ascending colon were numerous similar shallow ulcers of the size of a twenty-five cent piece, with irregularly scalloped edges. In the sigmoid flexure and rectum were formed, putty-colored, dry fecal masses. *May* 26, 1887.

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37. *Specimen of diphtheritic membrane from the tonsils.*

Exhibited by Dr. G. E. de Schweinitz.

Dr. Walter L. M. Ziegler sent me these specimens for microscopical examination, with the following history:

A young child was suffering with a severe attack of diphtheria of the fauces, which were well covered with buff-colored membrane. This was especially collected upon the tonsils. During a violent fit of coughing this curiously shaped mass, which as you see is a complete cast of the surface of the tonsil, was expectorated. It is irregularly convex above and has incurved fringed edges. The under surface is concave, uneven, and pitted. These irregularities show its attachments to the depressions and crypts of the tonsil. On the following day in a second fit of cough-

ing, an exactly similar mass was rejected. Inspection of the throat afterward showed two large excavated ulcers in the region of the tonsils, so deep as almost to look as if the glands themselves had been enucleated. A microscopical section of this membrane shows that its structure is entirely composed of rows and collections of darkly stained extravasated leucocytes, between delicate slightly granular film fibres. Some sections stained by Gram's method failed to show any micrococci.

June 9, 1887.

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38. *Two specimens of melanotic hypertrophy of the filiform papillæ of the tongue (black tongue).*

Exhibited by Dr. S. Solis Cohen.

I have the pleasure of presenting to the Society, two specimens, which, so far as I am able to ascertain, are without fellow in this country. The disease commonly denominated by some term equivalent to the English "black tongue" (*lingua nigra, nigrities linguæ, la langue noir, schwarze zunge*), was first described by H. Hyde Salter, and has been elaborately studied by French observers, especially by Raynaud and by Dessois, who described a specific causative microbe; the latter making futile attempts to cultivate the organism upon his own tongue. Within late years, English writers have reported a number of cases; and the most recent instance which I have seen recorded, is that studied by a German specialist, Shech, of Munich. The only case on record in this country is the one observed by me at the Philadelphia Polyclinic, in 1884, and alluded to by J. Solis Cohen in his article upon "Glossitis Parasitica," in Pepper's *System of Medicine*, as similar to those of Jonathan Hutchinson and other English observers, in which no microbe other than the ordinary organisms of the mouth could be demonstrated.

The specimen from this patient, a syphilitic negro child, is now under the microscope. The other specimen which I present, is from the tongue of an adult dyspeptic, who came under my care at the Medical Clinic of Jefferson College Hospital, in July, 1885. In illustration of the infrequency of the affection, I might say that in the latter clinic during my years of service as assistant and as Chief of Clinics, some 15,000 patients are recorded on the books, each of whom was told in pursuance of ordinary routine, to protrude the tongue; yet, but this one case was encountered in all that number. The appearance of the tongue is quite different from that of a tongue simply discolored by food or medicine, etc. The dorsum is covered in greater or less extent by a hairy over-

growth, black in color, more or less irregularly distributed in patches, usually connected one with the other, and largest and most frequent in the middle. In my first case there was one patch in the middle; in the other, four patches, occupying respectively the base, the middle, and the lateral borders of the dorsum, not, however, extending quite to the edges. Whatever be the site of the process, however, desquamation is likely to take place from the periphery toward the centre, with reappearance at varying intervals. Most of the recorded cases were quite chronic and were not amenable to treatment. The appearance of the tangled mass of black filaments in a well-marked instance, has been aptly likened to that of a field of wheat thrown down by the wind. Sometimes the patients complain of the roughness, or of heat, dryness, or other uncomfortable sensations; but, as a rule, they pay little attention to the affection, which is accidently discovered on medical inspection of the tongue for other reasons. The specimens I present show pigmentation of the imbricated epithelium of the over-growth (filiform papillæ); in one, greatest at the base, in the other, at the apex. Dr. Formad kindly examined them with higher powers than I have, and likewise failed to find any except ordinary bacteria—though, of course, it was rather late in the history of the specimens when he looked at them. This is also the experience of Shech, the latest observer. *June* 23, 1887.

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39. *Macroglossia.*

Exhibited by Dr. H. R. Wharton.

The patient from whom this specimen was removed was a girl, æt. 7 years, and the hypertrophy of the tongue had existed since birth, but it had gradually increased in size, and at the time of operation a large portion of the organ remained constantly protruded from the mouth.

The anterior portion of the tongue was removed by a V-shaped incision, and the resulting wound was closed by sutures. The child did well after the operation and the result was entirely satisfactory as regards the diminution of the size of the organ.

Hypertrophy with prolapse of the tongue or macroglossia, is generally a congenital affection, but it is also said to originate from inflammatory causes. The increase in the bulk of the organ may arise from hypertrophy of the mucous membrane of the muscular substance, or of the bloodvessels, or lymphatics. I think most modern pathologists are inclined to class this growth as a lymphangeioma. I also present a drawing of a baby, æt. 5 months, a patient in the Children's Hospital, who is suffering from the same affection. *June* 23, 1887.

III. THE VASCULAR SYSTEM.

1. *A case of general atheroma.*

Exhibited by Dr. J. H. Musser.

The friends of the patient from whom these specimens were removed, had him admitted to the Presbyterian Hospital, under the care of Dr. Richardson, on account of failure in his mental and physical powers. They had noticed that for a few years he was slightly "gone in his head," and that quite recently his mental incapacity was remarkable. At the same time, the past month, he lost flesh and strength.

When admitted, he could not give an account of himself. He was dull and apathetic, and readily lost the thread of a discourse; in fact, a connected conversation was impossible. Neither headache nor vertigo was complained of, however. It was seen that he was sallow in appearance, emaciated, and weak, and that œdema of the limbs, extending later to the scrotum, was present. He ate well, withal, and did not suffer from any dyspeptic symptoms; his bowels were sluggish; the only complaint of the patient was of dyspnœa, which occurred in paroxysms, without any physical signs in the heart or lungs to warrant such suffering. On physical examination, the lungs were found normal, the heart hypertrophied. The first sound was strong, the aortic second loudly accentuated. The apex beat of the heart was in the sixth interspace outside of the nipple line; its impulse was strong and heaving. The "pipe-stem" radials were readily felt, rigid, and of high tension. The urine was scanty, and contained a small amount of albumen, but no casts.

During his stay in the hospital (three weeks) the paretic condition became more pronounced, the paroxysms of dyspnœa continued; his speech became incoherent, and his ideas confused. One week before death he lapsed into semiconsciousness, which was attended with a low muttering delirium. In his helpless condition he fell out of bed, after which accident coma set in, resulting in death in twenty-four hours.

The patient, æt. 63 years, had been intemperate. His family history was good; his occupation, a weigher.

It was difficult to decide upon the cause of some of the symptoms during life. The dyspnœa was believed to be due to uræmia. The

general condition of the patient pointed to cerebral softening. It may be stated, by the by, that vomiting was quite common in the final week of stupor, but was not urinous in odor. The urine was very scanty in this period; a sufficient amount for testing could only be obtained by the catheter. The temperature was subnormal (chart lost). It seemed, therefore, that uræmia occurred as an epiphenomenon in the course of cerebral softening, which was due, no doubt, to atheroma of the blood-vessels. It may be remarked that at no time were there present any local paralyses, any changes in the pupils, or alterations in special senses.

The autopsy was held with interest, and revealed some unusual changes. On removal of the calvaria, a fresh blood clot was observed on the anterior half of the right cerebral hemisphere, and an excess of fluid (serum) was found in the ventricles. Undoubted softening of the brain, most marked in portions of the corpora striata, the medulla, and the pons, was present. The bloodvessels presented the most marked changes; all of those composing the circle of Willis were atheromatous, as well as most of the branches to their finest ramifications, detectable by the naked eye. In five places aneurisms were seen; one at the junction of the anterior cerebral and anterior communicating, one on the posterior communicating, one on each of the middle cerebral arteries, and one at the junction of the posterior communicating and posterior cerebral. They were from the size of a large bird shot to a pea. The walls of two of them were so thin as to be almost transparent. The arteries were tortuous. There was no thrombus in any vessel, and the source of hemorrhage could not be found. Portions of the pia mater and brain structure were put in hardening fluid to examine for miliary aneurisms.[1]

An examination of the remainder of the arterial system revealed a general atheroma. The aorta in its entire course, the coronary arteries, the cœliac axis and its branches, the gastric, splenic, and hepatic arteries, the mesenteric and renal arteries, were highly atheromatous; many of them exhibiting fatty and calcareous changes.

The valves of the heart were normal, save a slight patch of atheroma on a leaflet of the mitral. This orifice was incompetent, and the left side showed dilated hypertrophy. The entire heart, free from clots, weighed eighteen ounces.

The kidneys were highly granular, exhibiting scars and granulations, and a contracted cortex. The liver was fatty, the lungs were the seat of hypostatic congestion. Spleen normal. Hour-glass contraction of the stomach was found, but otherwise it was normal.

September 4, 1885.

[1] The specimens were lost through an attendant's carelessness.

2. *Small aneurisms of arch of aorta; compression and perforation of trachea; death from suffocation.*

Exhibited by Dr. W. Osler.

Dr. Osler exhibited the specimen, and gave the following account of the case. Patient, an Englishman, æt. 32 years, had come to Philadelphia on his way to Colorado, as he had been advised to winter there by his English physicians, who suspected the existence of lung disease. He had been in Australia, where he had lived a very active life. Had had a chancroid, and had taken alcohol in excess. For nine months before leaving England he had a cough, much worse at times. He came to the hospital August 31st, complaining of weakness and severe cough. Examination of the chest revealed no special areas of dulness, but many râles, mucous and sibilant, in front and toward the bases. At times the cough was very rough and hard, and there was much wheezing. There was irregular fever; the temperature on 14th reaching 102°. Remedies had very little influence on the cough. On the 17th he was found in a condition of stupor, and it was thought that he possibly had taken morphia, but the next day he was brighter. On the 21st there was blood with the expectoration. On the 24th he seemed as usual through the day, though ever since the attack on the 17th he had looked bad. Toward evening he again became very heavy and drowsy, and he was suspected to have obtained whiskey. The breathing became much embarrassed, and he had great difficulty in expectorating the mucus. At midnight he seemed sleeping very naturally. At 6 A.M. he was found dead. The face was suffused and cyanotic. From facts ascertained afterward, it seems that he had secreted a bottle of whiskey, of which he had taken largely.

The autopsy showed the lungs to be crepitant, the bronchial tubes filled with thick mucus, not blood-tinged. Anterior margin of the middle lobe of the right lung, and a margin of the lower of the left, were airless, and the bronchi dilated and filled with very thick purulent exudation. On slitting up the trachea, the tube, just above the bifurcation, was greatly narrowed by the projection of a hemispherical tumor, at the lower part of which was a small opening, through which blood exuded. The heart was not enlarged; valves healthy; aortic semilunar a little thick. Sinuses above the valves dilated. Entire arch of aorta widened, and the intima atheromatous. On anterior surface, just at pericardial attachment, was an aneurismal dilatation the size of

a walnut. The orifice of the innominate was also dilated. On the posterior wall of the transverse portion was an orifice not as large as a quarter dollar, leading into a sac the size of a large walnut, which directly compressed the trachea, and had eroded its wall. The sac was filled with firm clots. Just below the orifice of the subclavian were two shallow pouch-like dilatations, not lined with clots. The other organs presented nothing special.

Dr. Osler remarked that as he had not examined the case, he could not speak of the physical signs; the general symptoms were evidently those of severe pulmonary trouble. The compression of the trachea had caused the intense inflammation of the tubes, with exudation of thick mucus, and no doubt the fatal termination was due to plugging of the bronchi with secretion. The whiskey would, of course, hasten the suffocative process. *October* 8, 1885.

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3. *A comparison of the changes in arteries after ligature, and in the ductus arteriosus and umbilical arteries after birth.*

Read by Dr. J. C. Warren.

After the ligature of an artery in continuity the earliest changes noted, are the formation of the thrombi within the vessel, and the development of a mass of inflammatory tissue or callus around the point of ligature externally. No perceptible cell action can be observed on the inner wall with low powers during the first week, although, under favorable circumstances a proliferation to a limited extent of the endothelial cells near the point of ligature can be seen with high powers, and occasionally a few wandering cells may be found to have penetrated the walls of the vessel at the same point.

In the second week the bundle of fibres of the adventitia, which were surrounded and held by the knot, have been absorbed, and the two ends of the vessel retract slightly from one another leaving the ligature imbedded in and partly disintegrated by the granulation cells. The walls of each portion appear to form a complete cul-de-sac, and it looks at this time as if the healing process were complete, but it can hardly be said to have more than begun, as the vessel has not yet passed through what may be considered as the first stage of healing. The beginning of the second stage is marked by an unfolding of the ends of the vessel, the walls separating somewhat after the manner of the opening of a bud, which permits the entrance of a considerable quantity of the granulation

tissue. A disintegration of the thrombus follows, and we have now a fully developed external and internal callus, a small fragment of clot still protecting the latter from the current. The ligature may be totally disintegrated and absorbed, or it may have become encysted, or finally it may have created a small abscess about itself which has discharged the fragments of thread through a sinus opening externally. The second stage is now completed when the internal growth has reached the neighborhood of a branch. After this we have an absorption of the callus, which, as in fracture, is only a provisional structure, and eventually the two ends of the vessel are found held together by a slender cord of varying length. The walls of the vessel are slightly separated at each end by a cicatrix consisting of connective tissue externally, inside of which is another layer consisting largely of unstriped muscular fibre, the surface being covered within by a new endothelium. The cicatrix is always pierced by a vessel which terminates in a number of capillaries ramifying in the cord. We have here a scar made up of three layers resembling closely the three coats of the vessel.

In the large vessel of amputation stumps we have a somewhat different series of changes. Soon after the ligature, the end of the vessel may be seen imbedded in granulation tissue, and containing a thrombus of varying length. By the second week there is a marked change in the interior extending for some distance above the point of ligature; probably to the first large branch or to the origin of the vessel. Examined several months later, when the healing process has been completed, the vessel is found to be present in the shape of a cord running from the first large sized branch to the cicatrix of the stump. On laying open this cord the walls of the vessel are found preserved, the interior being filled with new tissue, leaving spaces occupied by one or more vessels. There has been a process resembling that known as obliterating endarteritis, by which the calibre of the blood channel has been narrowed to an extent to adapt it to the diminished blood supply. In this obliterating tissue we find comparatively large vessels with new coats consisting of an endothelium, an elastic membrane, and also a new media. An erosion preparation would best represent the condition of the arteries of the stump at this time. The main artery would, after giving off its largest branches, break into a spray of smaller vessels, no one of which would predominate. A comparison of these two modes of healing, with the changes seen in the arterial system after birth, shows certain resemblances in the two processes.

The ductus arteriosus about the time of birth, differs considerably from the structure of the aorta and pulmonary artery. The media is much

thicker than in either of these vessels; it is thrown into irregular folds, which are increased at the time of birth, and help to narrow its calibre. The outlines between the different layers of the wall are less marked than in other vessels. The lamina elastica is indistinct and in places apparently wanting; the media consists chiefly of longitudinal layers of muscular fibres, a few circular bundles existing in the outermost layer. A few weeks after birth a greater portion of the walls of the ductus undergoes hyaline degeneration; the outer or circular fibres of the media alone remaining, these being reinforced apparently by a growth from the media of the large vessels. At this time there is an active growth of long spindle-shaped cells with staff-shaped nuclei at the edges of the media bordering on the opening into the aorta; there is also a moderate thickening of the interior. Eventually the hyaline tissue becomes absorbed and is replaced by a ligamentous band of fibres which become continuous at either end with the media of the large vessels. At the aortic end in a longitudinal section, we see the media slightly separated at the point of the cicatrix, and between them and also continuous with them, are the longitudinal fibres of the ligamentum arteriosus. In this ligamentous tissue, and between the edges of the media, are numerous new muscular cells; nearer the surface is a layer of thickened intima, which in the aorta has not only connective tissue in it, but also a deep musculo-elastic layer. In the centre of the depression marking the site of the cicatrix, a small vessel is given off which penetrates into the axis of the ligament, where it either looses itself in a capillary network or becomes continuous with a similar vessel coming from the pulmonary artery. We have here conditions closely resembling those which have been described as existing in the cicatrix of a large artery after ligature in continuity, namely, the slightly separated ends of the media, between which lie the fibres of the ligament connecting it with the pulmonary artery; a new intima and a new growth of muscular cells, and finally a central arteriole. The only point of difference is the preservation of a layer of circular muscular fibres, which form an outer wall to the ligamentous band, a much needed support at a point exposed to great tension. Further protection is afforded by the oblique insertion of the ductus into the aorta, a valve-like action of the overhanging edge of the aorta diminishing the pressure upon this particular point.

At birth the umbilical artery, or that part usually called the hypogastric artery, the portion within the abdomen, at its origin from the internal iliac is a vessel of considerable size, being, in fact, a continuation of the upper portion of that artery. At its termination in the umbilical wound it has greatly contracted, and is filled with clot for a distance of

about one inch. The outlines of the various walls are not as distinct as in other vessels, and the elastic lamina is for the most part wanting. No special change is seen in the elements of the walls of the vessel except a slight accumulation of endothelial cells near the apex of the thrombus. A few weeks later there is a distinct growth on the inner surface of the vessel up to its point of origin; the terminal portion having undergone a hyaline degeneration and obliteration for a short distance. The vessel has greatly contracted throughout its entire length, and its calibre is further diminished by the growth in its interior. Cross-sections taken from the superior vesical artery in adult life, show the media as a thick wall out of all proportion to the size of the vessel, and consisting not only of its original wall containing longitudinal muscular cells interspersed with elastic tissue, but also an inner circular row of cells which are provided with a well-formed elastic lamina. It seems probable that the greater portion of the hypogastric artery has been preserved, the ligamentous band which extends to the umbilicus consisting of the obliterated extremity of that vessel, much elongated during the process of growth.

The series of changes which occur in the hypogastric artery after birth are closely analogous to those seen in the main trunk of an amputated stump. A slight portion of each vessel is destroyed, both retract and are attached to the terminal cicatrix by a band of fibrous tissue; both remain as pervious vessels, with thickened coats and narrowed calibre. In both, the process is not unlike that seen in the so-called obliterating or compensatory endarteritis. "Arteritis" hardly seems a term applicable to the changes taking place in normal arteries after birth, nor can the alterations which have been developed through the whole length of a large vessel, extending a considerable distance from the original seat of inflammation, be strictly regarded as of an inflammatory nature. May not the obliterative growths found to exist simultaneously in terminal arteries in widely removed portions of the body of the same individual also be regarded not as of inflammatory nature, but rather as a secondary and formative process, closely connected with disturbances in the mechanism of nutrition, designed to adapt the vessels to a diminished blood supply?

Dr. S. W. Gross said, in view of the fact that Dr. Warren's teaching seemed so directly opposed to that of other observers, he would like to have some points cleared up. He would, therefore, ask if Dr. Warren held, first, that instead of the external coat where it is included in the ligature, sloughing and coming away with the ligature, the pressure of the ligature sets up an irritation which causes the adventitia to return

to its embryonic state with a reconversion, after the ligature has cut through this to connective tissue; and, second, whether the repair of arteries was brought about by the ingrowth of the cicatrical tissue, which he likened to provisional callus, together with some proliferation of the muscular cells of the media.

Dr. Randall said that, as the result of careful study of the subject, he had always found the clot present at first, and that it was honeycombed even in the first few days by the contraction of its fibrin, through the spaces thus formed the blood seemed to be circulating. Cells, either original white cells or of endothelial origin, occupied the walls of these cavernous spaces and seemed to sheath them. The red cells early melted down into a homogeneous mass, furnishing the framework upon which the reparative tissue was built. The "plastic clot" of some observers he had not seen—the new cells being distributed throughout the old clot as well as upon the vessel walls and not specially collected in the immediate neighborhood of the ligature. The obliteration of the vessel was accomplished by the growth and contraction of the trabeculæ of new tissue built upon the remains of the original blood-clot. Even close to the ligature he had not found the vessel walls greatly altered, merely showing a great increase in the nuclei and in the number and size of the vasa vasorum, the lamina elastica being distinct, and, as a rule, intact. Toward the end of the first month the new tissue was largely spindle-cells, but careful staining had given no suggestion of muscular tissue. But, not having carried his study beyond the first month, he had no data upon this subject, since the development of muscular tissue is claimed to occur only at a later stage.

Dr. Formad was inclined to favor the views of Dr. Randall. The observation of Dr. Warren, that the new-formed connective tissue played the most important part, was to him entirely novel. He thought it probable that pressure upon the artery from without, with consequent diminution in its lumen, might bring about a condition more analogous to the growth of fœtal life than to inflammation.

Dr. Mears remarked that we are liable to be confused in considering this subject by the presence of the blood in the vessel. So far as the process of healing after ligature is concerned, we may consider only the structures which enter into the formation of the vessel wall, which is complex in character and composed of connective, muscular, elastic, and endothelial tissues. Dr. Warren has given us a very clear demonstration of cicatrization as it occurs in these structures after application of the ligature.

Dr. Tyson said that the most novel feature to him in Dr. Warren's

observations was the part played by the muscular tissue. This apparent increase in the muscular tissue he was inclined to believe was really only a proliferation of intermuscular connective tissue. He was becoming more and more convinced that there was only one kind of inflammation, the interstitial. He feared that he might himself mistake a proliferating connective tissue with spindle-cells for muscular tissue. It is true that new-formed capillaries and arterioles have muscular walls, but the development of these seemed to him to be a slower process. He had expected to find a process of repair in the ductus arteriosus different from that in an ordinary artery. This process he had expected would be a true endarteritis, for it seems that the conditions of closure here are rather different from those in the ligaturing of an ordinary artery and in the umbilical artery, where we have also ligation practised.

Dr. Warren, in closing, said, in cases where there is much breaking down and little repair, there might be a sloughing of the external coat, yet in his experiments this part of the arterial wall did not slough, but was simply absorbed by the granulation tissue, as is the ligature itself in some cases. In the normal condition even the elastic lamina is not a perfectly continuous plate; he did not refer to these breaks, however, but to numbers of little ruptures incident to the pulling out of the wound of an artery in the act of ligature; however, this need not necessarily occur. He had not attempted to identify anything like muscular tissue early; this could not be recognized till we had a permanent cicatrix formed. He had carefully eliminated all sources of error, and it seemed to him that in almost all cases there was a considerable number of these muscular cells. The layer was not always as well marked as in his diagrams, especially was this true of human cases, but these had all been in alcohol for some years; of the fresh specimens he had selected only those in which he had considered the process complete. In a specimen which he had here to-night, through an opening in the lamina elastica, cells could be traced from the muscular layer, those in the inside resembling exactly those outside. These facts, together with the fact that we have normally a few muscular cells inside of the lamina elastica, led him to believe in the proliferation of the muscular tissue. The disintegration of the blood-clot is accomplished by granulations growing inward from the callus, there being two sets of bloodvessels, one in the granulations, the other (blood spaces rather) in the clot itself; these unite about the end of the first or second month.

October 22, 1885.

4. *Aneurism of the ascending aorta, perforating the chest wall and forming an enormous external pulsating tumor in the right pectoral region.*

Exhibited by Dr. J. C. Wilson.

Mary M., æt. 74 years; widow; Irish; formerly a domestic, but for many years an inmate of the "Outwards" as a pauper, was admitted to the medical ward in the Philadelphia Hospital, June 16, 1885, on account of a hard lump in her breast. Upon her admission to the hospital, the tumor had attained large dimensions. It occupied the whole subclavicular and much of the mammary region of the right side, extending from the margin of the sternum to the anterior maxillary fold, and from just below the clavicle to the fifth intercostal space. It was irregularly circular in outline, with abrupt margins, and with a maximum elevation in the central portions of about five to six centimetres. The upper or clavicular margin was deeply notched, and from the angle of this notch a shallow furrow extended downward and slightly to the right, dividing the upper surface of the tumor in two lobes, one of which pointed toward the sternal notch, the other toward the shoulder. At several points the surface was marked by slight circumscribed elevations or bosses, two of which, namely, one in the sternal border and one in the upper and outer or acromial border were of a brownish-blue color and much less resistant on palpation than the surface elsewhere, which was, however, compressible at all points upon moderately firm pressure. To the eye the surface of the tumor appeared to be continuous with the mamma, but palpation discovered the lower margin above the base of the fifth rib, well-marked, abrupt, and continuous with the circular border, as elsewhere defined. The greatest vertical measurement, from margin to margin, over the convexity of the tumor, was about twenty centimetres; the greatest transverse measurement about twenty-two centimetres; the longest oblique diameter, from the border nearest the shoulder to the lowest point of the sternal border, was twenty-four centimetres. The tumor was everywhere the seat of a strong, expansile, heaving, systolic impulse. The skin, as well as the deeper tissues surrounding its face, was very tender to the touch. There was no external sign of inflammation. The heart was displaced slightly downward and to the left. Its impulse was of moderate force. There were no murmurs having their point of greatest intensity at or near the apex. At the base of the heart, over the second costo-sternal articulation, there was heard a loud double murmur. This bruit was distinct over a somewhat

limited area, which included a small part only of the tumor at its upper and sternal border, and was soon lost in the line of the carotid and subclavian arteries. It was faintly heard behind in the right supra- and interscapular spaces. At the lower part of the tumor auscultation revealed the presence of a harsh, low-pitched, but not very loud systolic bruit, which was restricted to a limited area. The upper and outer portions of the tumor beyond the limits indicated, yielded upon auscultation a distinct systolic shock, accompanied by a faint sound and wholly without murmur. The præcordial dulness was continuous with that of the tumor, and with flatness in the upper sternal region. There was dulness anteriorly on the right side beyond the limits of the tumor, and relative dulness on the right side posteriorly, less marked over the lower than over the upper lobe. Occasional mucous and subcrepitant râles were heard upon auscultation over the lungs. In other respects the physical signs betrayed no lesion of the left lung. The radial and carotid pulses, sixty-eight to seventy-two per minute, were alike in volume, time, and force on the two sides, with retardation as compared with the apex beat. The patient refused to have sphygmographic tracings taken. The pupils were alike and responded equally to light. There was no stridor. At rare intervals there occurred moderate paroxysms of dyspnœa. Cough was infrequent, and usually attended with scanty, thin, mucous expectoration. There had been no blood-spitting. Dysphagia was constant but not severe. The veins of the face and neck were not engorged. The temperature, taken in the axilla, was normal.

Diagnosis.—Aneurism of the ascending aorta, perforating the chest wall and forming a consecutive false aneurism.

Ten days after admission the tumor had undergone a considerable increase in size. The notch upon its upper border, and the sulcus described above as existing from this notch, had disappeared. The measurements, which had been made from and to fixed points, marked upon the skin with nitrate of silver, were found to have increased by two to three centimetres, and the mass was more protuberant. The pulsation had become exaggerated and at one or two points the discolored skin threatened to yield to the internal pressure of the blood. The tenderness was such as to render any attempt to support the tissues externally by strapping or a pad, impracticable. I determined to try electrolysis. The patient was with difficulty persuaded to submit to this treatment, which was, however, carried into effect on the 28th of June. The battery used was a modification of Stoeber's, with Flemming's zinc-carbon elements. The electrolytic needles were of steel, seven centimetres in

length, straight, elliptical in transverse section, 1.5 x 1 millimetre in diameter, insulated to within 7 millimetres of the point, which was very sharp, flattened, and heavily gilt. All the apparatus was supplied by Mr. Flemming, who also rendered valuable personal service at the operation. The patient was carefully informed of the nature of the procedure and cautioned against movement of any kind. Her usual quarter-grain hypodermic dose of morphia was administered beforehand, and the bed placed in a good light. Two needles connected with the positive pole were slowly introduced into the tumor at points some distance from its upper and lateral borders. They penetrated half their length, 3.5 centimetres, the points being freely movable within. A large electrode covered with a thin layer of fine-grained sponge, moistened and connected with the negative pole, was held firmly against the lower border; the battery was then put in action, five cells being at first employed and the number rapidly increased to ten, fifteen, and finally twenty cells, with a current strength, estimated by Mr. Flemming at twelve milliampères. With this number of cells, the current, as tested beforehand by placing the metallic tips in white of egg in a saucer, in a few minutes formed a small, dense, translucent coagulum around the positive pole, and a large, white, opaque, loose clot, the size of a marrowfat pea around the negative pole. The patient suffered much pain upon the introduction of the needles and at each increase in the number of cells. An attempt to use twenty-three cells caused suffering so intense that it was at once abandoned. The current of twenty cells was kept up thirty-five minutes; at the end of this time there was no appreciable diminution in the force of the pulsation, nevertheless, the sufferings of the patient were so great that it was thought best not to prolong the application. The needles were withdrawn slowly and with a rotary movement. A few drops of blood followed the one nearest the shoulder. The points of both were much eroded. For twenty-four hours cold water compresses were constantly applied to the tumor.

June 30. It was noted that there had been less pain than before the operation, and the pulsation seems less forcible.

July 18. Less pain and no increase in size till within the last four days. There is rapid extension of the mass at its axillary border; skin much discolored, bluish, and very sensitive; patient obliged to lie with the arm extended, to avoid touching it; wall at this point very thin; pulsation much stronger than elsewhere.

20*th*. Electrolysis was performed for the second time. As there was little apprehension of danger from embolism, it was determined to con-

nect the needles with the negative pole, in order to secure at each point as large a coagulum as possible without regard to its texture. The needles were introduced; one into the thin-walled upper axillary border, one into the upper sternal border, and one into the lower sternal border. The large sponge-covered electrode was held firmly against the lower and outer border of the tumor. Twenty cells were used from the beginning. At the end of twenty-eight minutes the pulsation throughout the tumor was much diminished; in thirty-three minutes the needle at the upper sternal border had ceased to move with the cardiac rhythm, that at the lower sternal border moved but slightly, while the excursion of that in the upper axillary border was much restricted. In forty-five minutes the movement of the needles had almost ceased. The *séance* was continued one hour. In the case of one needle only, that at the upper axillary border, was the withdrawal followed by bleeding. Here a little jet followed, which was, however, easily controlled by a compress, with the loss of not more than one fluidounce of blood. A cold water dressing was used as before. No inflammatory action supervened. The pulsation again increased after some hours, but was never so violent as before, nor did the tumor undergo further increase toward its axillary border, although there was some general increase in its bulk up till the time of the patient's death, which occurred from exhaustion on the 8th of September, some weeks after the ward had been transferred to the care of my colleague, Dr. Neff.

Autopsy, by Dr. E. O. Shakespeare, nine hours after death.—External appearances: Slight yellowish tinge of upper extremities; noteworthy appearance, that of large, rounded, prominence of right mammary gland and supramammary region. Its vertical diameter measured twenty centimetres, its transverse twenty-two centimetres; its most prominent point was eight centimetres above the level of the ribs. It extended transversely from the sternal border to the anterior axillary line, and vertically from a line just below the clavicle downward twenty centimetres. The skin over this prominence was not discolored, except at two points near the middle of its sternal border. The mass was moderately firm to the touch.

Thorax: Left pleural cavity normal, with the exception of moderately firm adhesions over the posterior portion of the lower lobe of lung. Of the left lung the upper lobe was normal, the lower consolidated posteriorly. The lower right lobe was normal; the middle lobe was very firmly attached by its upper and anterior surface to the wall of the chest and to the upper lobe, it was crepitant. The right upper lobe

was compressed, and was firmly adherent to the chest wall, so that it was torn in the attempt to remove it. Pericardial sac obliterated by old but not very firm adhesions. Heart not opened. The right anterior wall of the thorax, together with the hemispherical tumor, was removed with the heart and vessels attached, for further examination. It was found upon entering the finger into the ascending portion of the aorta, that it was the seat of aneurismal dilatation, and that there was a large aneurismal sac to the right.

Liver slightly enlarged; its cut surface brownish-red and friable. Spleen normal. Kidneys normal in size, capsules easily stripped off, surface granular. Upon section cortex seemed to be of unusual thickness and of pinkish-gray color.

A further examination, which I made at a late date, discovered the heart to be slightly increased in size; its muscle-substance normal; mitral leaflets sound; aortic leaflets thickened and crumpled. The aorta was extensively atheromatous, and was somewhat dilated in all its diameter, from the sinuses of Valsalva up to and including the transverse portion, with a large sac springing from its right wall, and forming an intrathoracic aneurism somewhat larger than the heart itself. The sac had greatly compressed the upper and middle lobes of the right lung, and had contracted very firm and dense adhesions to the surrounding structures. It had also, by pressure upon the ribs, caused displacement with crowding and extensive erosion of the second, third, and fourth ribs, the second and fourth being widely separated, and the third entirely absorbed for a considerable portion of its extent anteriorly. The wall of the sac had given way at the point of adhesion to the chest wall, forming an oval opening much smaller in its diameter than the aneurism itself, by means of which the blood had found its way beneath the muscles and fasciæ of the chest wall and formed the external or consecutive false aneurism, which was so striking a feature of the case. The intrathoracic aneurism contained but a small quantity of laminated clot. The external tumor was densely filled with firm laminated clot, in which, however, at the time of my examination, some weeks after death, no traces of the foci presumably caused by electrolysis could be discovered.

This case belongs to the group in which traumatism plays an important part in the etiology of aortic aneurism. The pain, which was the principal and most characteristic subjective symptom, was first felt shortly after the violent fall, in which the chest was struck against a block of wood. It is usual to ascribe the lesion in these rare cases to the sudden strain brought upon the walls of the artery, in consequence

of the effort made by the patient in falling to save himself, rather than to any direct injury to the arterial coats. It appears to me that in this case, the blow, leading, as it must have done, to forcible compression of the aorta, at the moment already filled, by the sudden effort to avoid the fall, greatly increased by its direct effect the already excessive arterial pressure, and so caused the walls to yield, not in the direction of the blow, but laterally. Old age and alcohol had, without doubt, rendered the arterial coats peculiarly incapable of bearing such a sudden strain. Scarcely less worthy of note than the enormous size of the aneurism both inside of and outside of the chest wall, is the almost complete absence of pressure signs. This is to be explained by the direction taken by the sac from the right antero-lateral aspect of the ascending aorta, and affords a striking example of the immunity from pressure which the important structures of the mediastinum may enjoy in thoracic aneurisms of even exceptional size. *February* 11, 1886.

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5. *Malignant endocarditis.*

Exhibited by Dr. T. S. K. Morton.

The patient from whom this heart was removed was an Italian, who spoke no English, which, together with his very short period of life after admission, rendered impossible the elicitation of any history.

He was found in an unconscious condition upon the street by a policeman, and removed to a station-house, where he quickly revived, but soon was taken with a succession of fainting seizures, and was brought into the Pennsylvania Hospital about 3 A. M. His symptoms upon admission were those of rupture of compensation in a diseased heart. Auscultation proved this to be true, for a loud, musical, double murmur was heard over the mitral area, while a feeble aortic regurgitant sound was also discernible. Under appropriate stimulation the cardiac rhythm was restored, and he slept quietly until morning. Another examination revealed nothing more, save symptoms of disorded cerebration—for which he was ordered to bed and to be watched. In the afternoon, however, he suddenly arose from bed, kept the nurse back by throwing at him whatever he could reach, opened a window, and jumped a distance of three stories, thereby sustaining a fracture of the base of the skull, from which he died some ten hours afterward.

The necropsy showed all of the organs to be healthy, except the brain and heart. The former presented chronic meningitis, both lepto- and pachy-, and other unmistakable evidences of insanity, while the latter presents, to a marked degree, the results of a former malignant endocarditis.

As is most common, so in this case, the mitral leaflets, and their connections and surroundings, have borne the brunt of the inflammatory process. The cusps of this valve are connected by organized inflammatory material, which, in fact, involves all of the surrounding structures, and travels along the chordæ, and into their papillary attachments, thickening, shortening, and greatly distorting them. The anterior flap is greatly thickened and tied down; the posterior is likewise enormously thickened, tied down, and contains several large vegetations with pea-sized foci of calcification. The organ is hypertrophied throughout, the tricuspid valve much dilated, admitting four fingers and the thumb, and the aortic leaflets are greatly thickened and much fenestrated, as are also those guarding the pulmonary artery. No other abnormalities are present.

March 11, 1886.

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6. *Aneurism of the larger cerebral arteries.*

Read by Dr. W. Osler.

Aneurisms of the branches of the circle of Willis play an important part in the history of cerebral hemorrhage, ranking next to miliary aneurisms of the nutritive arteries. They are certainly more common than the statements of text-books would indicate, and unless a careful dissection is made they are very readily overlooked. Abstracts of twelve cases are appended, nearly all of which occurred at the General Hospital, Montreal. In eight, the aneurisms had burst and caused fatal hemorrhage; in four, they were found accidentally, and had not produced any symptoms.

A majority of the patients were young or middle-aged individuals. One was a child of 6, another a lad of 17, and a third a man of 20. In five the age was about 40. The early age at which they occur has been noted by several writers. Thus, of seventy-nine cases collected by Coats,[1] there were forty-two between the ages of 10 and 40. The

[1] Glasgow Medical Journal, 1873

case here reported, occurring in a boy of 6, is the youngest of which I can find any note. Eight were males and four females. The arteries involved were: Left internal carotid, Case I.; right Sylvian, Cases II. and V.; left Sylvian, Cases III., V., IX., and XI.; basilar, Cases IV., VII., and VIII.; anterior communicating, Cases VI. and X.; and anterior cerebral, Case XII. The aneurisms ranged in size from a small pea to a large cherry. With the exception of Case IV., they were sacculated, and communicated with the lumen of the vessel by an orifice smaller than the circumference of the sac. In Case V. there were two aneurisms, one on either Sylvian artery. In Case III. the aneurism was surrounded by thickened meningeal tissue; in the others the sac was free. In seven cases the hemorrhage was chiefly meningeal, and the laceration of brain substance was slight. In Case III. the hemorrhage was altogether into the substance, which, from Coats's account, seems not uncommon. The extravasation was usually basic, and beneath the arachnoid; the amount of blood considerable, except in Case I. In Case X. the hemorrhage extended along the right optic nerve and appeared as a subconjunctival ecchymosis.

In Cases III., VII., VIII., and IX. there was heart disease; in Case VIII., ulcerative endocarditis. In Cases I., V., VI., VII., IX., and XI. there were atheromatous changes in the branches of the circle of Willis.

Embolism, endarteritis, and atheroma are the chief causes of aneurism, and the cases in this series afford illustration of each. Although it was suggested by Ogle,[1] Church,[2] and others that embolism played an important part in the production of aneurism, the evidence was not very conclusive until the publication of Ponfick's observations in 1873.[3] In several of his cases, the connection of the embolus with the aneurism was very clearly demonstrated. Of the cases here recorded, four were associated with heart disease, but in only one, Case III., was the condition suggestive of the previous occurrence of embolism. In this patient, a lad of 20, with aortic valve disease, the aneurism projected directly into an oval cyst with reddish-brown contents, and there can be little doubt that here, on a former occasion, a lesion of the vessel, most probably embolic, had occurred. Ponfick supposes the dilatation results from a weakening of the wall due to direct contact with the embolus. Recently, I have had an opportunity of studying an aneurism of the renal artery which had developed on one of the primary branches, and, indeed, involved to a slight extent the bifurcation. There were

[1] Medical Times and Gazette, 1866

[2] St. Bartholomew's Hospital Reports, vol vi.

[3] Virchow's Archiv, Bd. lviii

ulcerative endocarditis and a large infarct in the lower end of the righ kidney, with considerable fibroid change about it. The vessel going to this part was obliterated and fibroid in the greater part of its extent, but for one-fourth of an inch from the main division it was dilated and aneurismal. The walls were grayish-red in color, softer than normal, and the coats could be readily separated. Here the aneurism had developed on the proximal side of the obstruction in consequence, apparently, of interference with the nutrition of the coats of the vessel.

In the other cases associated with heart disease, there did not appear to be any trace of previous embolic lesion in the affected vessels. In six cases there were atheromatous changes in the vessels of the circle of Willis, and I think this process stands in more frequent connection with the formation of these aneurisms. Patchy atheroma, with fatty changes in the intima, is very common in the larger arteries of the brain, and I have met with instances in which the branches of the middle cerebral arteries were chiefly or alone involved. The occurrence of so many of these cases under the age of 40 has been urged in favor of the embolic theory, but atheroma, particularly in isolated foci, may occur in the young. A weak spot in one of the large cerebral vessels would be specially liable to yield, as the pressure in these branches is so directly communicated from the aorta.

In Case IV. the dilatation was due to local endarteritis, which was possibly associated with recent syphilis. There were subendothelial proliferation and infiltration of the media with leucocytes.

The clinical history of cases of cerebral aneurism is necessarily meagre, as they rarely cause symptoms prior to rupture. In six of the eight cases in which this took place, death occurred rapidly. In Case I. the hemorrhage was small, and the patient lived about six weeks; in Case XI. the duration of life after the rupture was ten days.

Case I. *Aneurism of left internal carotid.*—Female, æt. 53 years, admitted, under Dr. George Ross, December 24th, with headache and debility. Three days before she had had an epileptiform convulsion, followed by drowsiness, and in the succeeding forty-eight hours there were four other seizures. There was no paralysis, but the chief symptom was a rambling delirium, from which she could be roused, and then would answer intelligently. Urine albuminous. She remained in this state nearly five weeks. On January 25th the right arm was flaccid. Insensibility supervened, and death about six weeks after admission. The post-mortem examination showed atheromatous arteries at the base of the brain, and on the left internal carotid artery, just before its division, a sacculated aneurism the size of a hazel-nut, which communicated with

the lumen of the vessel by an orifice one by one and one-half lines in diameter. The sac contained a firm decolorized clot. In the course of the middle cerebral artery there was recently effused blood, beneath which the brain substance was lacerated to a slight extent. There was an atheromatous patch on the anterior segment of the mitral valve; otherwise the heart was healthy.

Case II. *Aneurism of right middle cerebral artery.*—Mrs. R., æt. 40 years, married, five children, patient of Dr. John Bell, was found speechless in her bed on May 29th. She had been a healthy woman, but had suffered with vertigo, and of late her memory had failed. There was left-sided hemiplegia, gradually deepening insensibility, and death occurred on the night of the 30th. At the autopsy, slight meningeal hemorrhage was found at base and over convolutions of right side. The right Sylvian fissure contained a large clot, and the convolutions bounding it were considerably lacerated. At the main bifurcation of the right middle cerebral artery was an aneurism the size of a bean, about half an inch in length and a quarter of an inch in breadth. At its under surface was a rupture with a ragged orifice. There was no atheromatous change in the vessels at the base. No heart disease.

Case III. *Aneurism of left middle cerebral artery.*—A. R., æt. 20 years, a small, but well-built man, died suddenly on the evening of the 25th of March, and the body was brought to the hospital. No history could be obtained of any previous illness. Brain, on section, presented a large clot on the left side, which involved the lenticular nucleus, internal capsule and part of thalamus, and reached almost to the convolutions of the insula. On carefully tracing the vessels in the left Sylvian fissure, one of the vessels was closely adherent in the angle between the insula and the parietal convolutions. The artery appeared to enter an oval mass the size of a large cherry, which, internally, was in direct contact with the clot, and on slitting up the vessel it expanded into a small aneurism the size of a pea, which occupied about one-third of the oval mass above referred to. The wall of the aneurism presented a rupture four millimetres in length. The chief part of the oval mass was made up of a cyst with firm walls and reddish-brown, pulpy contents. No communication existed between this and the aneurism, but at one point the connection of the aneurism and the cyst was very rough and fibrous. A branch was given off just below the aneurism, which looked as if it had been formed at the fork of a vessel. Arteries of the circle of Willis were not atheromatous. Heart hypertrophied; fusion of two of the aortic cusps; no vegetations.

Case IV. *Aneurismal dilatation of left vertebral and first part of basilar arteries; endarteritis.*—James B., æt. 36 years, found dead in his bed. Eighteen months before had had secondary syphilis. Extensive coagulum at base of brain from optic commissure to medulla, and extending along the vessels into the fissures and filling the fourth ventricle. The left vertebral and first portion of the basilar much dilated, and in the latter vessel, close to its origin, there was a shallow dilatation, with a small perforation in the centre. The intima was smooth, but in places presented opaque atheromatous areas, which, about the centre of the basilar, very materially reduced the lumen. The carotids and Sylvian vessels normal. Heart healthy. No other regions of arterial disease.

Case V. *Aneurisms of right and of left middle cerebral arteries.*—Male, æt. 55 years, patient of Dr. Arthur A. Browne. Ill for eighteen months with obscure brain symptoms. Vessels at the base very atheromatous, and just beyond the first division of the left middle cerebral there was a sacculated aneurism the size of a pea. It had not ruptured. On one of the main branches of the right middle cerebral artery there was a second small irregular dilatation. In the left hemisphere there was an old apoplectic cyst, in the vicinity of which were numerous miliary aneurisms. No valvular disease of heart.

Case VI. *Aneurism of anterior communicating artery.*—Mrs. G., æt. 40 years, died suddenly in a shop, and was brought to the General Hospital. No history of previous illness. Clots in region of longitudinal fissure, and a uniform sheeting at the base from olfactory bulbs to cord, entirely beneath the arachnoid. One or two spots of atheroma on basilar and middle cerebral branches. Careful dissection of the circle of Willis revealed a small aneurismal pouch projecting from the anterior communicating artery, and on its under surface a slit-like rupture 1.5 millimetres in length. The sac was smooth-walled, very thin, and presented a spot of atheroma near the orifice. Heart normal. Aorta atheromatous. Kidneys a little granular.

Case VII. *Aneurism of basilar artery.*—J. S., over 75 years of age. Death from thrombotic softening in left hemisphere of brain. Vessels at the base atheromatous. An aneurism the size of a large pea was connected with the basilar artery, and lay imbedded in a shallow fossa in the pons. The walls were thick, and did not contain thrombi. Heart hypertrophied. Aortic valves incompetent. Pericardium adherent.

Case VIII. *Aneurism of basilar artery.*—M. W., male, æt. 43 years. Had had syphilis. Admitted with pneumonia, and developed ulcerative endocarditis. An aneurism six by five millimetres projected from the upper wall of the basilar artery, about its centre, and had formed a bed

for itself in the pons. It did not contain clots. Branches of circle of Willis not atheromatous. Heart a little hypertrophied. Recent ulcerations on aortic and mitral valves.

Case IX. *Aneurism of left middle cerebral artery.*—Female, æt. 40 years. Died of pneumonia. Vessels of circle of Willis slightly atheromatous. A patch in the basilar narrowed its lumen considerably. Just before the first bifurcation of the left Sylvian artery there was a sacculated aneurism the size of a pea. The wall of vessel about it not atheromatous; no clots in interior. Heart hypertrophied; vegetations on aortic valves.

Case X. *Aneurism of anterior communicating artery.*—G. E., a lad, æt. 17 years, admitted December 18th in an insensible condition. Three months previously had an epileptic fit, from which he quickly recovered. For eight days past had had severe headache. He remained unconscious and died on the 23d. Ecchymosis of right upper eyelid and conjunctiva developed while under observation. Extensive hemorrhage at base of brain, involving meninges and extending along the anterior cerebral arteries upon the corpus callosum. On separating the orbital plates of the frontal lobes, and carefully removing the clots, an aneurism (measuring 10 by 11 millimetres) was seen occupying the longitudinal fissure. It was partially imbedded in the adjacent brain tissue, which was a little lacerated. On dissection, it was found to spring from the anterior communicating artery by a very small orifice situated close to the right anterior cerebral. The sac was full of dark blood, walls very thin, and presented a rent of 2 millimetres in extent at the lower part. Other vessels of the brain healthy. No heart disease.

Case XI. *Aneurism of left middle cerebral artery.*—Woman, æt. 62 years. Admitted in a semiconscious state, with right hemiplegia, which came on after a fit three days before. Death occurred on the seventh day after admission. There was extensive hemorrhage in the meninges of the left Sylvian fissure, and thin clots also at the base. The inner and anterior part of the left temporal lobe was lacerated. A small sacculated aneurism the size of a pea was found on the left Sylvian artery, just within the fissure. The walls were extremely thin, and the orifice of rupture large. The vessels of the circle of Willis were atheromatous. No heart disease.

Case XII. *Aneurism of anterior cerebral in longitudinal fissure.*—Boy, æt. 6 years, brought to hospital unconscious, with feeble pulse, pale face, eyes and head turned to right, and left hemiplegia. Death in six hours. He had fallen from a hay-loft three weeks before, but recovered rapidly from the effects. There was meningeal hemorrhage at base

and in longitudinal fissure. On separating the median surfaces of the hemispheres, and clearing away the blood, a small nodular projection was seen on the right side, about the middle of the gyrus fornicatus. This proved to be an aneurismal sac imbedded in the calloso-marginal fissure just where it turns vertically upward. The rupture was on the meningeal surface, but there was hemorrhage into and slight laceration of the contiguous portion of the brain substance. The arteries were not atheromatous, and the heart was healthy,

Our knowledge of the subject of cerebral aneurism may be thus summarized:

1. The rupture of aneurism of the large arteries is a frequent cause of cerebral hemorrhage in persons under forty years of age; in the experience of some pathologists, the most common cause.

2. The hemorrhage is usually extensive, and may be exclusively meningeal or mainly into the brain substance and ventricles. (Coats.)

3. The aneurisms are caused in a few cases by embolism, in a large number by atheroma and fatty change, and occasionally by acute endarteritis. *May* 13, 1886.

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7. *Aneurism of the aorta; sac filled with firm laminated clot; no physical signs on auscultation or palpation; pressure symptoms; death from acute pleurisy; autopsy.*

Exhibited by Dr. J. H. Musser.

The specimen presented weighs three pounds four ounces, and includes the heart and diseased bloodvessel, the adjacent vessels, nerves, and air-passages, and a portion of the sternum and ribs. The aneurismal tumor is intimately adherent to that portion of the sternum between the lower part of the first rib and the lower border of the third rib. It is in close approximation to the remainder of the bone above and below to the lower border of the fourth rib. It extends to the right of the edge of the sternum two and a half inches in the first, two in the second, and one in the third interspaces. The right lung overlaps it and is adherent at the lower part of the sac. The tumor extends beyond the left edge of the sternum one and two inches in the first and second interspace respectively. The left lung overlaps it slightly below the third rib.

The mass fills the superior mediastinal space. It grew backward and

toward the right, and in doing so pressed downward upon the right bronchus, causing the trachea to be pulled downward and to the right. Pressure was made anteriorly on the substernal veins, and posteriorly in the median line and to the right, on the trachea. The remaining structures were not pressed upon. The nerves, vessels, and œsophagus are seen, herewith, intact.

The tumor is the size of the head of a one year old child. It is irregular in shape and of a moderately firm consistence. It arises from the aorta two inches from the valves. The opening into the sac from the vessel is two and one-half inches long and two inches wide. The edges of the opening are clean-cut and atheromatous. The remaining portion of the aorta is atheromatous. It can be seen that fibrin in laminæ fills the entire tumor, which is solidified by these clots. The clot is excavated to the depth of an inch and a half by the blood current. This area is lined with delicate new connective tissue.

The heart is in close apposition by its base, to the lower surface of the aneurism. It was pushed downward and to the left. The apex was in the sixth interspace. The cavities of the heart contained ante-mortem clots.

The remaining organs of the body were healthy. A very considerable recent pleural effusion with some lymph was found on the left side; it was this that caused death.

As indicated in the heading, the physical signs of aneurism usually elicited on auscultation and palpation were absent. On inspection the trachea was observed deflected to the right, and the superficial subcutaneous veins of the upper left area of the thorax were enlarged. Percussion revealed an area of absolute dulness which corresponded to the measurements given above, although slightly lessened along the right edge of the tumor by the overlapping of the lung. A faint systolic murmur was heard in the right carotid. Bronchial breathing was heard between the scapulæ. There was no difference of pulses or pupils.

The patient did not suffer at any time from pain. Dyspnœa and cough were the only marked symptoms. They have continued three years, and were worse on exertion and at night. A tired sensation was observed by the patient in the breast in the early period of the disease. The laryngeal image, revealed by the glass, was normal. The cough and dyspnœa were undoubtedly due to pressure on the trachea. The patient was slightly emaciated. He denied syphilis but had some indurated glands. He was more or less intemperate. His age was forty-eight; he was single; his personal medical history was good, with the exception of an attack of rheumatism eight years ago, which continued, more or less, one year. Family history good. *November* 11, 1886.

8. *Anomalous anterior papillary muscle.*

Exhibited by Dr. W. Osler.

Anomalies of the smaller muscle-bundles of the heart are so rarely met with that the following example is worth recording. The specimen was obtained from a middle-aged woman, who died in the Philadelphia Hospital of uræmic convulsions. There were no heart symptoms.

Heart.—Heart is of medium size, right chambers not dilated, edges of tricuspid a little thickened. Left ventricle, walls slightly thickened. Mitral orifice admits two fingers; segments a little thickened. A curious anomaly occurs in the anterior papillary muscle; it is conical in shape, inserted by a broad base into the outer wall. It presents three main muscular divisions. The anterior, the largest, passes directly on to the anterior mitral segment, to which it is attached for a distance of nearly three-quarters of an inch, the apex reaching close to the insertion of the left coronary segment of the aorta. At point of insertion it is joined by a flat band to the muscle of ventricle below aortic ring. From the other two divisions of this muscle chordæ tendineæ arise. Also, from the abnormal portion three or four main cords spring. The aortic segments a little thickened; left coronary small. Aorta ascendens a little dilated and atheromatous. Muscle of left ventricle from fifteen to twenty millimetres in thickness; a little pale. Coronary arteries pervious. *November* 11, 1886.

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9. *Aneurism of thoracic aorta; perforation into left pleura.*

Exhibited by Dr. W. Osler.

Mabel W., æt. 22 years, was admitted to the Philadelphia Hospital complaining of pain in the abdomen. She had been dissipated and had had syphilis. I saw her the next day at 4 P. M. She was a medium sized well-nourished woman, anæmic, and looked as if she had been drinking; was extremely nervous and excitable. She complained of pain in the epigastrium and left side. I made a careful examination of the abdomen and thorax, without detecting anything abnormal. The heart sounds were clear. About 4 or 5 o'clock the next morning, she got out of bed complaining of agonizing pain in the abdomen and side, and in a few minutes fell dead on the bed.

Autopsy.—On opening the thorax, the mediastinum with the heart was pushed to the right by an extensive hemorrhagic effusion into the left pleura, amounting to several pounds. The lung was collapsed. When the pericardium was opened, it was noted that the apex of the heart was not pushed beyond the middle line, and the oblique position of the organ was retained in spite of the dislocation of the mediastinum by the large effusion.

Heart: Right auricle contained about ℥j of blood and clot. Left auricle and ventricle empty. On further dissection valves normal, the arch of aorta smooth, muscle substance pale and flabby.

Aorta: The arch was small, and the lining membrane healthy. Thoracic aorta looked normal until level of the ninth vertebra; here there was an opening on the posterior wall an inch and a quarter in length by a half inch wide. The contiguous parts of the aorta looked infiltrated and swollen. This orifice communicated with a sac full of clots, which lay immediately in front of the tenth and eleventh vertebræ, and projected into the left pleura, where it was closely united to the diaphragm. At the point of greatest prominence, there was a transverse laceration a half inch in length. The sac was about the size of an orange. The bodies of the ninth and tenth vertebræ were eroded, the latter most deeply. The cartilages were not involved. No changes of note in the other organs. *November* 11, 1886.

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10. *Aneurism of the root of the subclavian artery.*

Exhibited by Dr. J. C. Wilson.

The patient, a light mulatto man, deep-chested, well built; had been in good health till within eighteen months of his death. Before Petersburg a spent ball had struck him in the right side of the chest, the scar of which was still visible in the second interspace two inches to the right of the sternum. After the war he was a porter, lifting heavy weights continually, then a lamplighter. He had not had syphilis and had not used alcohol to excess. Eighteen months before his death he began to be slightly short of breath, with occasional attacks of pretty sharp dyspnœa, especially at night. In a few weeks his voice became husky and then was lost altogether. No laryngoscopic examination was ever made. During the whole course of his illness there was no pain. On examination, was found a strong heaving pulsation at the root of the neck, espe-

cially on the left. Dulness extended some distance to the right of the sternum as well as to the left, opposite the manubrium. In the suprasternal notch and over the manubrium there was a distinct pulsation. At the aortic cartilage, not conducted out into the arteries, was a short, low-pitched, rather harsh systolic murmur. The heart was displaced slightly downward, the apex beat being in the sixth interspace and a little to the left of normal. The left pulse was perceptibly feebler than the right. There was no difference in the pupils. Under rest and appropriate treatment he rapidly improved. Five weeks before the end his dyspnœa suddenly increased and became urgent, gradually bringing about his death. There had never been any spitting of blood.

Autopsy.—Heart slightly enlarged. No lesion of the mitral valve and none of the aortic, except a slight thickening of the leaflets, with, at one place, in the middle cusp, a small patch of atheroma. Aorta decidedly dilated and highly atheromatous. Just to the left of the point of origin of the left common carotid artery was an aneurismal tumor, presenting at first glance the appearance of a tumor of the aorta itself; on careful examination, however, this was found to be a tumor of the left subclavian artery. It was filled with a firm organized clot, and pressed upon the left common carotid artery, and also directly and somewhat forcibly on the trachea and left primary bronchus. The orifice of the subclavian was dilated, being fully three-quarters of an inch in diameter. *January* 13, 1887.

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11. *An anomalous tricuspid valve.*

Exhibited by Dr. W. A. Edwards.

The man from whom this heart was secured was admitted to my wards in St. Joseph's Hospital about three months ago. The most exacting symptom was dyspnœa and cardiac irregularity and palpitation; the man improved, and became a walking case, when suddenly, about ten days ago, he was attacked with orthopnœa, and died in a short time from asystole.

Autopsy, twelve hours after death.—Brain healthy. Sligh effusion into pleura and pericardium. Heart in an advanced condition of dilated hypertrophy. Aortic and pulmonary valves healthy. Mitral widely dilated, accounting for a systolic regurgitant murmur, which was heard at apex. An atheromatous patch was noted in the aorta just where the ascending arch became transverse.

The posterior leaflet of the tricuspid valve did not join its fellow, a space of about an eighth of an inch existing between the curtains of the valve. This is just at the "undefended space," and may be readily seen by holding the heart up to the light; it is, furthermore, the selective point for many congenital abnormalities, under which, readily, this valve could be properly classified. There was no pulsation either in liver or venous system. *February* 10, 1887.

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12. *Large phlebolith of long saphenous vein.*

Exhibited by Dr. W. Osler.

The specimen was obtained from G. H., an elderly man, who died in the Philadelphia Hospital of fatty and dilated heart. The tumor was noticed during life, but there is no note as to how long it had existed. There was no sign of a wound or of external injury, though the size and situation suggest that it may have resulted from traumatism.

Aorta smooth; common iliacs large; femorals present plates of atheroma. In Scarpa's space two inches below Poupart's ligament was a firm tumor the size of a large egg. On pressure it was elastic. Its relations on dissection were as follows: The femoral artery passed directly beneath it and was not involved. Femoral vein was free. Orifice of the long saphenous vein occluded about a quarter of an inch from orifice. The occlusion was at the tumor above mentioned, which occupied the upper end of the long saphenous vein. The vein was pervious from below, and entered the tumor directly at a distance of an inch and a quarter from the femoral vein. It seemed to terminate in the tumor by three or four small passages. On section, it presented externally a dense fibro-calcareous structure; the contents brownish, tolerably consistent, altered blood. *February* 10, 1887.

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13. *Rupture of the posterior papillary muscle of the left ventricle of the heart.*

Exhibited by Dr. W. Osler.

B. P., æt. about 70 years, a large-framed negro, was in Ward 6 of the Philadelphia Hospital, four months, with symptoms of mitral valve disease and heart failure. When I first saw him, about a month before

his death, the legs were swollen, and the urine scanty. There was orthopnœa, and slight effusion existed at right base. The apex beat was outside the nipple line; impulse forcible; and a systolic thrill could be felt in the apex region. There was a loud, rough systolic murmur heard well into the axilla. The pulse was irregular; superficial arteries very atheromatous. His history was not very clear. He had been a hard worker up to a few months before his admission to the hospital. His symptoms appear to have come on gradually, and throughout were those of mitral insufficiency. Three weeks prior to his death Cheyne-Stokes breathing came on, and persisted without intermission. During this time he was well enough to get out of bed for his dinner, and at this time he was repeatedly seen to wait for the urgency of the respiratory movements to pass away before taking a mouthful.

Body that of an elderly, well-built man. Œdema of legs, back of thigh. Abdomen contained a small amount of serous fluid. In thorax effusion into both pleural sacs. Pericardium contained a moderate amount of fluid. The pulmonary veins, arteries, and also the aorta, were filled with dark, firm clots. Right auricular appendix filled with an ante-mortem clot softened in the centre; several globular concretions in the neighborhood. The right auricle was much dilated. Tricuspid orifice admitted four fingers to second joint with thumb between. Cusps thickened; ventricle dilated and hypertrophied, muscular columns large. The pulmonary semilunar valves thin and clear. Left auricle dilated, walls somewhat thickened. Mitral orifice as seen from auricle not stenosed. Left ventricle dilated. Chamber circular. Anterior segments of mitral thin and natural looking. Chordæ tendineæ smooth, not thickened or shortened; plates of atheroma at the base of this segment. The posterior segment was normal at the part where the anterior chordæ tendineæ were attached. From the posterior papillary muscle four or five chordæ passed to the portion of the posterior segment next the aortic ring. A group of six or eight chordæ were attached on the middle and under portion of this segment, and their papillary ends were twisted and attached to a yellowish-brown portion, looking like the apex of a papillary muscle. This portion of the valve flapped freely, and could be pushed into the auricle. There were no vegetations. On the posterior wall of the ventricle, near the main mass of papillary muscle, there was a flat, slightly roughened surface, from which the small papillary muscle had been torn. The aortic segments were a little thickened, but competent. Both coronary segments fenestrated; calcification at attached margin. Aorta atheromatous; orifices of coronaries large; the arteries much calcified. Muscular substance of heart of good color.

Walls of the left ventricle were from a half to three-quarters of an inch in thickness. Weight of heart twenty-five ounces.

Remarks.—Careful microscopical examination showed the loose portion to which the central chordæ of the posterior valve were attached, to be undoubtedly the tip of a papillary muscle. Indeed, it represented one of the chief divisions of the muscle, torn off close to the heart wall. Here and there a muscle fibre could be seen imbedded in brown pigment and surrounded by fibrous tissue such as in elderly people so often supplants the proper tissue of the musculi papillares. The specimen is unique in my experience, and I do not remember to have noted an instance in literature. The absence of vegetations on the torn surfaces is interesting.

March 10, 1887.

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14. *Bicuspid aortic valve.*

Exhibited by Dr. F. A. Packard.

This specimen was removed from the body of Mary L., æt. 44 years, suffering from arterio-capillary fibrosis.

Heart shows slight hypertrophy of left ventricle, with slight dilatation. At aortic orifice there seem to be but two cusps, both having calcareous vegetations upon them. The partition between the right and left coronary cusps is merely represented by a slight ridge of what looks like new-formed connective tissue, but this is possibly the poorly developed representative of the division normally present. Hydrostatic test shows incompetence, the vegetations so filling up the orifice as to render inconspicuous, what without them would be a very marked deficiency. Teased preparation of heart muscle shows marked fatty degeneration.

Liver fatty and cirrhotic; nutmeg appearance present on section. Portal vein markedly dilated.

Kidneys slightly congested. Bloodvessels stand out plainly in cortex, which is somewhat diminished in thickness.

Lungs show a low grade of emphysema. Other organs normal.

March 10, 1887.

15. *Fibroid heart.*

Exhibited by Dr. E. T. Bruen.

These specimens are very interesting on account of the rarity of the heart lesion. The left ventricle is dilated and hypertrophied. The mitral and semilunar valves are normal. The aorta is covered at many points with atheromatous plaques. Upon the septum and involving the columnæ carneæ of the right ventricle, a large fibroid plaque can be seen. The new tissue crosses the ventricle, and is firmly attached to the right ventricular wall. This new tissue formation is situated about two and a half inches above the apex of the right ventricle. On section, it has been found to penetrate the muscle of the heart to the depth of several lines, but does not pass completely through it. On the exterior surface of the ventricle indistinct cicatrices can be seen. The pericardium elsewhere is normal. The liver presented marked evidences of perihepatitis and cirrhosis, weighing only three pounds. The kidneys were of normal size, both showing evidences of increase in the connective tissue at the cortical portion, and the capsules were adherent in places. The general fibrosis and the heart lesion seem referable to syphilis as a cause. Death was due to dropsy and heart failure.

In the *Transactions of the London Pathological Society*, vol. xxv. p. 65, will be found an interesting series of cases of fibroid disease of the heart, presented by Dr. Fagge. *March* 24, 1887.

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16. *A series of cases of death resulting from injury to the heart.*

Exhibited by Dr. H. F. Formad.

Case I.—Stab wound. The knife had passed through the left lung and anterior wall of the left ventricle near the apex, severing some of the chordæ tendineæ. The man lived for thirty-eight hours. Around the stumps of the severed chordæ tendineæ and in the wound, an organized clot had formed. There was some purulent pericarditis with blood in the pericardial cavity.

Case II.—Gunshot wound. The bullet had entered the muscular tissue of the left ventricle, but had not penetrated its cavity. The man died more than a week after the injury, of purulent pericarditis.

Case III.—Gunshot wound. The bullet had penetrated the sternum and anterior wall of the ascending aorta, dropping thence into the heart. The man lived a few hours.

Case IV.—Gunshot wound. The muzzle of a rifle had been placed against the chest and the rifle fired. The heart was torn completely open from base to apex, and the columnæ carneæ torn off. Death was instantaneous. Here the injury to the heart was due to the explosive effects of the gases generated rather than directly to the bullet.

Case V.—Stab wound. The knife pierced the left lung and the wall of the right ventricle. The man lived five hours. The pericardium was found distended with blood. *April* 14, 1887.

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17. *Pericarditis.*

Exhibited by Dr. F. P. Henry.

Patrick Mc. G., æt. 29 years, laborer, was admitted to the male medical ward of the Episcopal Hospital, March 31, 1887, suffering with an attack of acute articular rheumatism, confined entirely to the wrist-joints. He had the appearance of a man who had been drinking hard, but positively denied it. His expression was wild, and he was very restless and nervous, with slight delirium when asleep. On making inquiry from a friend, it was found that he had been drinking hard for the past year, very seldom sober. His delirium got worse while his rheumatism improved. A few days later he developed congestion at the base of both lungs, with pleurisy of the left side. The heart sounds were feeble and distant, but no cardiac friction sound was audible over the præcordia. His condition gradually grew worse, he complained of no pain, and died at 10 P.M., April 10th.

At the autopsy, the cavity of the pericardium was found filled with serous fluid and its walls covered with thick shaggy lymph.

May 12, 1887.

18. *Heart from a case of death from ether.*

Exhibited by Dr. H. F. Formad.

The specimen was from a man, æt. 50 years, about whose condition previous to the inhalation of the ether, nothing was known. The patient had partially recovered from the effects of the ether when he died, two hours after the ether was stopped.

The left lung was collapsed and adherent, and the heart pulled up out of place. The walls of the heart were thin and friable, and the right ventricle notably dilated. The kidneys were in a state of advanced parenchymatous degeneration. *May* 26, 1887.

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19. *Heart from a case of acute ulcerative endocarditis.*

Exhibited by Dr. F. A. Packard.

K. W., white, æt. 26 years, was admitted to the medical ward of the Pennsylvania Hospital, with the following history: Had never had rheumatism; was entirely well until two months before admission, when she caught cold and had a cough, but was not seriously ill until three weeks before admission, when she caught fresh cold and had fever. Took to bed two weeks before admission on account of cough, fever, and chilly sensations. Seven or eight days before admission she first noticed a rapidly developed shortness of breath, cough with blood-streaked expectoration, swelling of feet, great palpitation of heart, and pain in epigastrium. These symptoms steadily grew more marked until her admission, when she presented the following symptoms: Orthopnœa, sweating, irregular hurried pulse, labored, noisy, and rapid respirations, apex beat displaced to left of nipple, heart dulness increased in area, with blowing systolic and diastolic murmurs heard all over chest, but most clearly at position of apex and at aortic area. There was also a limited blowing systolic area at tip of xiphoid cartilage. There was a marked degree of tenderness in epigastric region. Albumen and pale granular casts were found in urine. Patient had marked sweating and irregularly high temperature. Patient died two days after admission to the hospital.

Post-mortem examination showed pericardium distended with turbid serum.

Heart large, globular in shape; ventricles filled with both ante- and post-mortem clots; weight of heart fifteen ounces. Heart muscle pale, fatty, and friable; walls thickened; cavities much dilated. Tricuspid orifice admits four fingers easily. Pulmonary valve apparently normal. Mitral valve leaflets thickened, orifice enlarged. On under surface of aortic leaflet of mitral valve is an ulcerated orifice communicating with the sinus of Valsalva back of the left coronary leaflet of aortic valve. Aortic valve leaflets all thickened. On the edge of the left coronary cusp is a mass as large as the tip of the little finger, with a rough surface covered by clotted blood. Just below this cusp is seen an ulcerated point surrounded by thickened endocardium dissected up from heart muscle, and through this opening is seen a probe passed through the fistulous tract that forms a communication between the mitral funnel and the sinus of Valsalva. Beside the opening in the sinus of Valsalva already described, there are two others making communication between the left ventricle and the sinus of Valsalva. *May* 26, 1887.

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20. *Dilatation of heart.*

Exhibited by Dr. F. A. Packard.

Removed from the body of Sarah B., colored, æt. 50 years. Weight of heart sixteen ounces. Apex formed by left ventricle. Walls of all chambers very thin. Muscle fatty and friable. All chambers enormously dilated. Walls much thinned out. Interventricular septum almost as thin as writing paper. No valvular outgrowths, thickenings, or contraction. Tricuspid orifice easily admits four fingers and thumb. Mitral orifice admits four fingers. Foramen ovale wide open. In wall of left ventricle are seen numerous pearly patches of fibroid character. Globular thrombi present at tip of left ventricle and in left auricular appendix. *May* 26, 1887.

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21. *Pericarditis; dilatation of aorta; gall-stones.*

Exhibited by Dr. F. A. Packard.

Specimen removed from the body of Mary J., colored, æt. 47 years. Pericardium enormously distended with fluid holding large flakes of creamy, yellow, stringy lymph. The fluid measured about one quart.

On anterior surface of heart is a fibroid patch, rough on its surface, and projecting fully a quarter of an inch from the surface of the heart, and covering an area of the size of a silver dollar. On the parietal layer of pericardium is a corresponding patch. Visceral pericardium lustreless and faintly granular to the touch. No valvular lesion present in heart. At root and extending as far as the orifice of the innominate artery the aorta is greatly distended, the inner surface showing signs of beginning atheroma, the cavity being large enough to contain a large base-ball. Gall-bladder packed with stones, one large round one being situated at orifice of gall-bladder into the cystic duct.

May 26, 1887.

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22. *A case of spurious aneurism of the innominate artery.*

Exhibited by Dr. H. A. Hare.

Annie D., æt. 17 years, a weaver, came to me complaining of pain in the chest and in the arm, one year ago, and dated its onset to three months before. She had no history of syphilis, but one of repeated attacks of acute articular rheumatism, to which her father and younger brother were also subject. Her mother and remaining brothers and sisters were well and hearty. There was no history of traumatism or scarlet fever.

On inspection, there was to be seen an egg-shaped protrusion in the suprasternal notch, very expansile and bulging at each systole of the heart. The dilatation extended well up into the innominate artery for over an inch from its point of origin. Pulsation was strongly marked in all the vessels of the neck. The apex beat of the heart was in the sixth intercostal space, about one inch to the left of the nipple. There was no distinct thrill over the præcordial space, but it was well marked in the supra-sternal notch and over the dilated portion of the innominate artery. On auscultation over the swelling there were heard a rough and a soft murmur, accompanied by a bruit. Over the aortic orifice was heard a double murmur—a short, sharp, and rough systolic, and a long, softer, and more blowing diastolic. These murmurs were very distinct over the sternal notch, becoming intensified as the ear was carried down the sternum, reaching their maximum at the ensiform cartilage. Both murmurs could be heard in the vessels of the neck, the diastolic feebly. There was a typical " water hammer " pulse, twenty-seven to the quarter-

minute, with marked difference in the volume of the pulse in the radial arteries to the touch, and as registered by the sphygmograph, the right falling behind the left.

There were cardiac dilatation and great hypertrophy, but no dysphagia, dyspnœa, or loss of voice. She also at one time complained of symptoms of abdominal aneurism, some of which were present under physical examination. She was found dead in bed about fourteen months after she was first seen by me.

The diagnosis during life was innominate aneurism with aortic dilatation. The post-mortem examination revealed that there was absolutely no lesion whatever of the bloodvessels, either as regards rupture of a coat or dilatation; the only condition found being one of great elasticity. There was no abdominal aneurism. The bloodvessels in the cadaver were not dilated but of normal calibre, and had simply been distended during life by the arterial pressure. *May* 26, 1887.

IV. THE ORGANS OF RESPIRATION.

1. *A case of perichondritis of the cricoid cartilage.*

Exhibited by Dr. L. Jurist.

Mr. H., æt. 37 years, married, of good habits and good family history, was treated by my friend Dr. De Young, for a mild attack of bronchitis, limited to the upper lobes, from September 9 to 12, 1885, inclusive. He again appeared on the 19th, complaining of huskiness and some cough, without expectoration. There was no dysphagia, no pain, no external swelling, but considerable fever. The following day he remained much the same; his cough, however, inducing vomiting. On the 21st, improvement began; cough diminished, the voice cleared up, and on the 22d, after a friendly chat with his physician, he expressed the desire to follow his usual clerical work. This he was cautioned not to do, but he disregarded the admonition, and left for his place of business the following morning. He remained at his books until evening, and on his way to take a street-car home, noticed dyspnœa marked enough to necessitate resting several times while walking a block. At 7.45 P.M. he was suddenly seized with a suffocative attack. Dr. De Young, living in the immediate neighborhood, was at once summoned, and finding his circulation good, and no cyanosis present, considered his symptoms due to spasm. Shortly after, he appeared easier, and expressed a desire to sleep. Having been hastily sent for, Dr. De Young and I reached him at 9 P.M., and found that he had just ceased breathing. An immediate laryngotomy and artificial respiration failed to restore him.

Post-mortem examination, fifteen hours later, limited to the thoracic cavity. Lungs crepitant and pale, the right bound down by old adhesions. Heart normal, pale, filled with fluid blood and clots. The larynx and trachea, removed by being drawn into the chest cavity, are presented for examination. Contrary to expectation, laryngeal œdema is conspicuous by its absence. The laryngeal surface of the epiglottis is crimson in spots; there is arborescent injection of both aryteno-epiglottic folds. Both ventricular bands bright red. Vocal bands, particularly the right, reddened and infiltrated. About one-third of an inch below the right vocal band there is a gangrenous patch of mucous membrane about one-

third of an inch in diameter. On the left side, at a point exactly corresponding to this, a very much smaller spot of gangrene is also noticeable. The trachea is slightly red in patches. On splitting the larynx from behind (which should not have been done) the knife slipped into an irregular cavity on the right side, from which about a half teaspoonful of thick pus exuded. This cavity extended under the gangrenous patch above mentioned, and showed a carious condition of the cricoid cartilage, and loss of perichondrium. There was also a well-marked plate of ossification. On the other side, a similar but much smaller cavity was found, by carefully following a minute opening in the gangrenous mucous membrane.

The incomplete post-mortem permitted, together with the absence of a more extended history of the patient, make the determination of the etiology, as well as the exact mode of death in this case, almost impossible. The condition of the larynx and the lungs excluded tuberculosis. Acute primary perichondritis, running a rapid course, or a late syphilitic infiltration of the cricoid perichondrium, seems to be the only explanation. In the absence of the patient's history, and an ante-mortem laryngoscopic examination, this point must remain undetermined. As there was neither extrusion of any portion of the cartilage, nor an amount of pus sufficient to cause suffocation, death was apparently due to prolonged laryngeal spasm. This case is interesting not only from the pathological standpoint, but as illustrative of a possible, though rare, form of "sudden death." *September* 24, 1885.

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2. *Aspergillus from the lung.*

Exhibited by Dr. W. Osler.

The specimen was sent by Dr. Rogers, of Denver, Col., with the following history: "Mrs. H., æt. 29 years. In Colorado for two years. Mother died of phthisis when patient was eight months old. She is a robust and vigorous woman, and states that she has always enjoyed good health. When seventeen years old she began to cough up bodies similar to the accompanying one, but smaller, at intervals of about three months—never more than one at a time, and it was generally unbroken. The bodies have gradually increased in size, and the intervals have become shorter, until now the attacks recur every two or three weeks. The bodies, as you may see from this one, are now of the size and shape of a small, white

bean, and present a soft downy outgrowth on all sides but one, which is flattened and smooth and has a small opening in the centre which leads to a little cavity. The color is of a light gray, but dark on the attached side. On examination, I found it to be a vegetable fungus, and send it to you for more accurate determination. Mrs. H. has no cough, except from one to three days before one of these bodies is expelled, and it is finally brought up by a very violent cough, and she describes it as coming with some force into the larynx against the vocal cords, where it sometimes lodges until another cough expels it. Shortly before it comes up she has a 'husky,' obstructed sensation on taking a deep breath, but no hoarseness or marked discomfort of any kind. For a short time she experiences a disagreeable, musty taste. After its expulsion she has no further symptoms until the next attack. Her husband, who is a physician, has examined the lungs repeatedly, and can find nothing abnormal."

The specimen conforms to the excellent description of Dr. Rogers, and on examination is seen to be made up almost exclusively of the mycelium and spores of an aspergillus, most probably *A. glaucus*. There is no portion of lung or bronchial tissue with it, or any mucus or adherent cells.

Cases of so-called pneumonomycosis aspergillina are rare, not more than eight or ten cases having been reported. The literature is fully given by Furbringer, in Virchow's *Archiv*, lxvi. The majority of the specimens have been discovered post-mortem, in old cavities or spots of hemorrhagic infarction. The points of special interest in this case are: (1) the occurrence in a woman in apparently good health; (2) the remarkable duration of the affection; and (3) the recurring attacks of coughing which result in the expulsion of the fungus.

October 8, 1885.

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3. *Anomalous lung.*

Exhibited by Dr. F. A. Packard.

Right lung of negro, who died at the University Hospital. On the inferior surface of the lower lobe, somewhat posterior to the centre, is a tongue-shaped process of pigmented, crepitant lung tissue. The process is about two and a half inches long and is attached by a broad base to the lower lobe. The width of the process at its widest part is about two

inches, tapering to a point. The process rested on the dome of the diaphragm by its inferior edge, the external surface of the process being in apposition with the inner surface of the lower lobe of the lung.

Another lung, also in the body of a negro, was found with a process similar to the one shown, except that it was smaller and situated somewhat more anteriorly. *November* 12, 1885.

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4. *Larynx and trachea from a case of membranous croup, in which tracheotomy had been performed, showing an extensive deposit of false membrane.*

Exhibited by Dr. H. R. Wharton.

The history of the case from which this specimen was taken is as follows:

Mary L., æt. 4 years, was under the care of Dr. J. H. Lopez, for membranous croup. On August 5th her respiratory difficulty was so marked that he advised her parents to take her to the Children's Hospital for the operation of tracheotomy, but they disregarded his advice and postponed removing her to the hospital until the following day.

When I saw her a short time after her admission, she was suffering from marked symptoms of laryngeal obstruction. Her condition was so urgent, that I gave no ether, but exposed the trachea and opened it as rapidly as possible, dilating the edges of the tracheal wound with Golding-Bird's dilator before introducing a No. 2 tracheal tube. The opening into the trachea in this case was made quite high up, through the cricoid cartilage and upper portion of the trachea. As time was an element, this was rendered necessary by the extreme shortness of the neck and the large size of the isthmus of the thyroid gland; for unquestionably much time is saved by the high operation where these conditions exist. At the time of operation the smallness of the trachea was noted, as it only accommodated a No. 2 tube.

The operation was followed by immediate relief as regards the symptoms of dyspnœa, the child falling asleep before it was removed from the operating table to its bed. The case progressed favorably for nineteen hours, without any return of symptoms of respiratory obstruction, taking nourishment well and sleeping most of the time; at this time, after having a passage, the child fell back and died.

Post-mortem examination revealed a complete membranous cylindri-

cal cast of larynx and trachea, extending into the larger bronchi, as may be seen by examination of the specimen; there was also an ante-mortem heart-clot.

This case presents several points of interest:

1st. The immediate relief of the symptoms of dyspnœa which the operation afforded, in spite of the extensive deposit of membrane which must have existed below the point at which the trachea was opened at the time of the operation. The relief afforded here is, I think, analogous to that which is obtained where the operation is performed for impacted foreign body in the trachea or larger bronchi, in which marked dyspnœa often exists, and is relieved by opening the trachea even where it is impossible to remove the foreign body; showing, I think, in both conditions, a certain amount of reflex laryngeal spasm.

2d. The extremely small calibre and the shortness of the trachea for a child said to be four years of age, are remarkable; for even in children of two years or a little less, I have been able to introduce the next larger sized tube, No. 3, without difficulty, although in female children the comparative smallness of the trachea is generally noted.

3d. The extensive deposit of membrane in the trachea and larynx, a tubular cast of both existing and extending into the larger bronchi.

4th. From this condition, I think, a point of great clinical interest is developed, for had the wall of the trachea only been divided and a tube hurriedly introduced, it would have pushed the false membrane down in front of it and caused a fatal impaction below the end of the tube; this accident has occurred in a number of cases, and explains the death in one case that came under my immediate observation.

This unfortunate complication of the operation was avoided in this case by a free incision, which divided both trachea and false membrane, and by the use of Golding-Bird's dilator, which kept the tracheal wound dilated so that the trachea could be cleaned of any loose membrane and the tube introduced without difficulty. *December* 10, 1885.

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5. *Pneumonia of apex and meningitis.*

Exhibited by Dr. T. S. K. Morton.

The patient from whom the above specimens were obtained was a woman of dissolute habits, æt. 29 years, who was brought into the Pennsylvania Hospital on the ambulance, from a house of ill-fame in the

lower portion of the city. She was entirely comatose, and but the following brief history could be obtained:

Until five days before admission she had been perfectly well and supposedly healthy, when, after exposure she was shortly taken with a chill and pain in the lower portion of the right breast. Soon she became fevered and short of breath, and gradually worse up to the time of her admission. She was then found to be in a state of profound coma; when disturbed would utter incoherent sounds, as of low delirium. She was very rigid, and opposed, somewhat, any passive movement; when moved, the arms and legs sprung to their original positions. When the shoulders were raised the whole body had a tendency to rise up like a log. Muscular tremors and subsultus tendinum were very marked. The pupils were contracted to pin points. She lay perfectly flat and could not be aroused. The temperature was 104°, pulse 160, respirations 36. The lips were dry, cracked, and fissured, and blackened by dried extravasated blood. Tongue and mouth were dry and red. Stomach retentive. Examination of heart and abdomen was negative.

Lungs: Respiration very noisy—many tracheal râles. Left lung: large mucous râles, hyper-resonance and exaggerated respiratory sounds. Right lung: increased vocal fremitus and resonance at apex, while this area upon percussion was dull, with spots of tympany posteriorly. Throughout the lower portion of this lung fine crepitant and subcrepitant râles were heard; auscultation of dull area revealed metallic tubular breathing. The skin was hot and dry; face not flushed. She had neither cough nor expectoration, and the pulse, though rapid, was moderately firm and bounding.

By active antipyretic measures the temperature was soon reduced to 103.2°, at which point it remained for a few hours, when it again began to rise, and, in spite of most active antipyretic treatment, steadily increased, until at 8.30 the next morning 106° was reached, and maintained until death occurred at 11 A. M. During these last hours the pulse was 160, respirations 60. Much urine but no feces was passed involuntarily during the night. Toward the end, and while the fever was still at 106°, she was bathed in a profuse clammy sweat. The pupils were still tightly contracted when examined a few moments before death.

Necropsy, four hours after death.—Rigor mortis well marked; various evidences of former specific disease.

Brain: Dura mater in spots slightly adherent to calvarium. Superior longitudinal sinus contained a well marked ante-mortem clot extending its entire length, and at about its middle extending into and entirely occluding a large vein of the pia mater; this vein had ruptured about

an inch beyond, and had poured out a drachm or two of fluid blood beneath the pia mater. The pia mater was in a state of acute lepto-meningitis, which had gone on to suppuration in many spots. There was also an ante-mortem clot in the middle meningeal artery at its origin, with consequent congestion of the supplied arteries beyond. Considerable effusion existed in the ventricles.

Lungs: Right showed well-marked croupous pneumonia passing from stage of red into that of gray hepatization, involving the superior lobe; rest of this lung, save recent plastic adhesions between superior and middle lobes, normal. Left lung showed spots of atelectasis only.

Heart: Fenestration and slight thickening of valves.

Kidneys were in the first stage of parenchymatous inflammation.

Uterus and appendages: Double tubal obstructive dropsy; cystic degeneration of right ovary. *December* 10, 1885.

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6. *Pyo-pneumo-thorax subphrenicus.*

Exhibited by Dr. W. Osler.

W. S., æt. 24 years, was admitted to the surgical wards of the University Hospital on November 13, 1885, having fallen under the wheels of an engine. The left arm was crushed and he had a deep scalp wound. The arm was amputated at the upper third. For a week he had hæmaturia and he complained of a pain in his left side. Subsequently erysipelas developed in both arm and face. About three weeks after admission, signs of inflammation appeared in left infra-scapular region, indicated by a rise of temperature, dulness and feeble, blowing breathing, and he was transferred to the medical ward. The stump at this time had almost healed. Examination of chest revealed circumscribed dulness at left base, extending nearly as high as angle of scapula, and, laterally, to mid-axillary line. Tactile fremitus was diminished; on auscultation, feeble, blowing breathing, and on deep inspiration râles. Slight cough, very little expectoration. A septic pleurisy was suspected. The condition remained practically unchanged for several weeks, during which there was irregular septic fever. He complained at times of pain in the ilium and left side, particularly when he drew a deep breath. He soon began to spit up fetid pus, and in twenty-four hours brought up several ounces. It was concluded that a localized empyema had perforated the lung. On examination, tympanitic resonance, amphoric breathing, and metallic

râles were found low down in the postero-lateral region, beneath the ninth, tenth, and eleventh ribs, indicating pneumo-thorax.

The autopsy showed the existence of a large abscess behind the left kidney and descending colon, extending from the diaphragm to the crest of the ilium. The chief part of the abscess lay above the kidney, beneath the ribs, and in this region there was a distinct cavity, partially occupied by dirty-brown pus, similar to that which the patient had expectorated during the last two days of his life. Part of the diaphragm was in a sloughy condition, and two orifices, through each of which the point of the index finger could be passed, communicated directly with an abscess cavity in the lower lobe of the left lung. The pleural membranes of this part were greatly thickened, and there was a small localized empyema between the layers. There were areas of recent broncho-pneumonia throughout the other lobes. The left kidney was small, and presented at its upper part a distinct cicatrix, to which the capsule and adjacent tissues were strongly adherent.

The sequence of events in this case was, probably, as follows: Wound of kidney with bruising of tissue in lumbar region; subphrenic abscess; localized empyema, probably from contiguity with subphrenic abscess; perforation of diaphragm and lung, with discharge of pus; development of a subphrenic air-containing cavity which gave, in the lower and lateral aspects of the left side, the signs of pneumothorax.

Most of the cases of subphrenic pneumothorax have been in connection with perforation of the stomach, duodenum, or colon, but Sänger, in *Archiv der Heilkunde* for 1878, describes two cases, not unlike this one, following injury. The literature, together with the record of a case occurring with perityphlitis, is given by Dr. Gardner, in vol. ix. of the Canada *Medical and Surgical Journal.*

Dr. Carl Siler would like to ask Dr. Osler, in view of the fact that post-mortem there had been found a perinephritic abscess, and that early in the case there had been hæmaturia, whether he thought the abscess was due to the fractured kidney. He asked this, because in the case of a workman who fell in a fit and afterward had hæmaturia and pains in the loin, he had found in the urine blood, pus, a few kidney cells, and one or two casts, and on these slight grounds he had ventured a diagnosis of fractured kidney. Post-mortem examination showed this diagnosis to be correct.

Dr. H. R. Wharton had seen the case reported by Dr. Osler a short time after his admission to the hospital. In the light of the later developments he thought the case a very interesting one, as showing how a kidney lesion might easily be overlooked, when there were other grave lesions present. This man had a bad scalp wound and well-marked

symptoms of concussion of the brain, and a crush of the arm, which necessitated amputation. He was inclined to think the abscess was due to the kidney lesion. Mr. Morris, of London, in speaking of retro-peritoneal abscess, had described twelve cases in which abscesses had arisen after injury to the kidney, five of which afterward ruptured through the lung. Hemorrhage is as frequent and constant a symptom in contusion of the kidney as in rupture of that organ; in the latter condition the blood may clot in the ureter, occluding it, and the abscess go on developing without the urine furnishing any significant symptoms.

Dr. G. G. Davis suggested that another way to account for the abscess was, that it might have begun as a septic pleurisy and gone down. It could scarcely have commenced from laceration of the kidney, as this seems to have healed perfectly. He had seen, in two or three cases of pyæmia, a purulent condition about the diaphragm, and pus in the pleuræ and mediastinal tissues. Pus has a great tendency to diffuse itself, and hence might easily have gone down; we might, in these cases, learn much of their origin by a careful study of the symptoms, and Dr. Osler had said that here pain in the loin was not complained of.

Dr. H. F. Formad, through his office of Coroner's Physician, had become well acquainted with the previous history of the patient. He had been subject, for some years, to a cough, and was a hard drinker.

Dr. J. C. Wilson remarked that he had treated, two years ago, in the wards of the Jefferson College Hospital, a case of circumscribed empyema of the left side, in which the pus had broken its way downward and pointed externally, at the level of the second lumbar vertebra.

January 28, 1886.

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7. *Two cases of papilloma of the larynx.*

Exhibited by Dr. A. W. MacCoy.

Case I.—Ernest S., æt. 8 years, a native of Chester County, and a patient of Drs. Isaac Massey, of West Chester, and H. Darlington, of Concordville, was referred to me in July, 1884, for treatment for papilloma of the larynx. The child's parents gave the following history: One year and a half before coming under my observation they had noticed that the lad was suffering from a mild catrrrhal laryngitis, accompanied by a slight cough. A few months later, they noticed the lad's voice changing—becoming thin, shrill, and often breaking. This condition continued for six months, his voice coming and going. Nearly

one year afterward, his voice had become reduced to a mere whisper, and only with great effort could he raise his voice to a *hoarse* whisper.

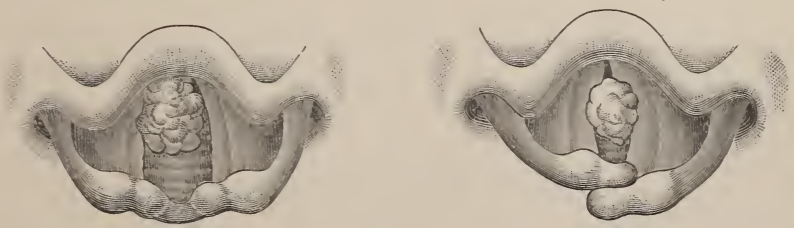
About this time the lad complained of oppression, and some pain in the chest. A few months before, his parents had first observed difficult breathing, but not until three weeks before I saw him had the attacks of dyspnœa become frequent or alarming. Severe attacks of difficult breathing occurred at night, and often lasted for hours, and suffocation appeared inevitable.

At his first visit to me his breathing was quite loud, stridulous, both on inspiration and expiration, more marked on *inspiration*. His face was pale and anxious; pulse soft, irregular, and rapid; inspiration and expiration laborious, giving the impression that suffocation might occur at any moment. The lad had lost flesh rapidly during the previous three months.

Laryngoscopic examination showed the lumen of the glottis greatly encroached upon by a grayish, pinkish mass, irregular in outline, completely obscuring the left vocal band. The mass appeared thick and piled up on the vocal band, overlapping and overhanging the free edge, and descending underneath the left vocal band. The right vocal band was *less* involved; a growth of considerable size appeared to spring from underneath its middle. There was, in addition, a *subglottic growth*, which on deep expiration was seen through the rima-glottidis filling up the entire chink, except a small space at the posterior portion. This growth appeared to spring from the anterior angle of the thyroid cartilages, just beneath the anterior commissure of the vocal bands. This mass was very freely movable on respiration—*inspiration* drawing it down into the trachea almost out of sight, while *expiration* forced it *up* into the chink. This condition of affairs produced a most novel effect—a constant bobbing up and down during respiration—and at the same time constituted a state of imminent danger to life. It was under such circumstances, demanding instant action, that I was called upon to decide upon the proper treatment. The child had already been examined by another laryngologist of this city, who refused to do anything prior to a tracheotomy. The parents were averse to subjecting him to what they deemed an additional risk, and were anxious to have an attempt made to remove the growth before tracheotomy should be resorted to. Accordingly, the growth *was* removed by the intra-laryngeal method without tracheotomy or previous training of the larynx, and necessarily without cocaine, as the operations were performed prior to its use. The several growths were removed at various sittings by Mackenzie's right-angled forceps (cutting and blunt) until the larynx and trachea were entirely

free from the papilloma. The tendency to recurrence was checked by the application of fused chromic acid. At the present writing the lad's voice is fully restored in volume and character, and the anatomical features of the interior of the larynx are intact.

Case II.—During the early part of April, 1885, F. W., male, æt. 28 years, applied to me for relief for a loss of voice. His history is as follows: In May, 1883, he first noticed that he could not sing low notes (B flat); his voice was uncertain, and often failed him; he had not noticed any cold. He could not sing bass (his customary part), but could easily sing in tenor register. This condition continued during the summer. In September, 1883, he began to notice that his speaking voice was growing hoarse and getting weaker; had a short cough. Continued hoarse all winter. In April, 1884, he went to a medical clinic in this city, where he was examined, and an opinion given that he had paralysis of the left vocal band. He spent the summer of 1884 at the seashore, his voice constantly growing thinner and weaker. Lost flesh, but appetite continued good; tried electricity. During the winter of 1884–85, his voice still continued growing weaker; finally, a stoppage in his breathing occurred when asleep, awaking him with a start, and a long, stridulous inspiration followed. Any exertion was followed by obstructed and difficult breathing. This state of affairs continued until his voice was reduced to a whisper, and a feeling of suffocation was often experienced. In April, 1885, he came under my observation. Laryngoscopic examination showed at once the cause of all his difficulty. The appearance of the interior of the larynx is faithfully portrayed by Dr. J. Madison Taylor, in the accompanying picture. The left vocal band and the



arytenoid were seen to be immovable upon phonation, remaining abducted; paralysis of the lateral crico-arytenoid and arytenoid muscles; the right vocal band was only partly visible, being obstructed in its middle and anterior portion by an overlapping tumor of irregular outline, and of a pale red color. It was quite bulky, and appeared to spring

from beneath the vocal band. On phonation, the growth in part appeared between the vocal bands, and prevented free movement of the right side. This tumor was first attacked with forceps, after a twenty per cent. solution of cocaine was applied. The larynx was unusually deep-seated, so to speak, and the ordinary right-angled forceps of Mackenzie were too short, only a small portion of the growth being torn off. No more of the mass being obtainable by forceps of the usual length, forceps an inch longer were made. These were more efficient in reaching the growth, but what was gained in length was lost in power, and while a considerable portion of the tumor was removed, no perceptible gain was made, as the papilloma seemed to grow as fast as it was removed.

After many attempts to use forceps of other shapes, guillotine, and snares, I abandoned all these, and began treatment with the galvanocautery point. The tumor had now grown to greater dimensions than when first seen. The larynx being fully anæsthetized by cocaine, a pointed electrode covered with rubber was easily introduced, and the point pushed well into the papillomatous mass; connection was then made, and after a few seconds the point was withdrawn, showing the mass in part a whitish color. Considerable shrinkage took place in the tumor, and no unpleasant secondary complications whatever followed. These sittings were continued until the mass was reduced to a mere thickened outline underneath the vocal band; this was finally removed. Patient's condition began at once, and continued, to improve; his voice improved as the growth grew less, and changed from a deep hoarseness to its natural tone. To-day his voice is fully restored, but not quite pure; it would be perfect, if it were not for the paralysis of the left vocal band. The right vocal band is normal in color and perfect in outline, no loss of substance being noticed anywhere. In phonation the right side overrides so much to the left that the immobility of the left is fairly compensated. *January* 28, 1886.

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8. *Diaphragmatic pleurisy; death from tetanus.*

Exhibited by Dr. J. H. Musser.

Patient admitted to the Presbyterian Hospital under the care of Dr. Woods. Notes by Dr. Taylor.

Male, æt. 20 years, white, single, driver, admitted April 12, 1886. Prior to admission suffered for two days severe pain in the left chest

near the diaphragm, increased by breathing. A small dry cough and some fever accompanied it.

On admission, short dry cough; pain as above; respiration 34, regular; pulse regular and of fair strength; temp. 103⅖°. Slight dulness at the extreme lower portion of the left lobe posteriorly with distant breathing. Turpentine locally. Quinia, gr. ij, every four hours. Carbonate of ammonia with liq. ammon. acet.

April 13. A. M. temp. 104°, P. M. temp. 104°. Not so much pain or cough. Davis's powder, gr. ij, and calomel, gr. ½, every two hours.

14*th*. A. M. temp. 102⅘°, P. M. temp. 100⅖°. It is noted he is much better and the beginning pneumonia checked. Patient cheerful and eats well.

15*th*. Morning temperature 98⅘°, evening, 98⅘°. Cough loose and rather painless. Sedative cough mixture ordered.

16*th*. A. M. 97⅗°, P. M. 98⅕°. Patient slept well. Bowels opened.

19*th*. Pain in the right chest. Evidences of commencing pleurisy at the base. No physical signs in the left side.

23*d*. Slight pneumonia, according to the physical signs, at the left base. No fever. Appetite better; patient cheerful.

25*th*. This morning stiffness of neck and locking of jaws with pain in the occiput. No fever. External stimulating liniments relieved the neck trouble.

26*th*. Worse again; 10 A. M. general tetanic convulsion. Chloral, gr. xl per rectum at 11 A. M. and 5 and 3 P. M. In the evening no relief, and ext. physostigmæ fl. by hypodermatic injections used without relief. Inhalations of ether and nitrite of amyl did not control the spasms. After twelve hours of spasms, at 11 P. M. death took place from heart failure.

At the autopsy the left lung was found adherent to the diaphragm over the larger part of its central area, and thence to the posterior portion. The lung was not adherent to the costal pleura, there was no effusion and no evidences of a recent pneumonia. The right lung was deeply congested along the posterior part of the lobe, but portions did not sink in water. It, also, was attached to the diaphragm by recent lymph, and a small area of adhesion between it and the thorax was seen at the lower edge of the lung posteriorly. The remaining organs of the body were healthy. The brain and spinal cord were examined and no lesions observed. By an oversight the phrenic nerve was not examined.

Remarks.—The case is remarkable on account of the excessive double diaphragmatic pleurisy without the symptoms usually attributed to this

disease. The complication of tetanus is also worthy of notice. In not one of the many cases analyzed by Donaldson (*Amer. Journ. Med. Sci.*) is this complication. It is possible that some pathological alteration of the phrenic nerve, as a neuritis, must be invoked to explain this unfortunate disaster. *May* 13, 1886.

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9. *A case of acute phthisis.*

Exhibited by Dr. H. W. Fisher.

Joseph F., æt. 27 years, single, stable boy, colored, was admitted to the Episcopal Hospital, March 21, 1886. In his exhausted condition we were not able to gain much information from him of a satisfactory nature as to his previous health, but subsequently through the politeness of Dr. Paxson, of Jenkintown, in whose employment he had been just before his admission to the hospital, I learned the following facts. He had for a long time led a roving life, having been employed in a hotel stable and travelled with a circus. About April, 1885, he had chancre followed by non-suppurating bubo and some weeks later by joint pains, ulcerated sore throat, etc. He also drank to excess. On July 28th he was employed by Dr. Paxson to take care of his horses, carriages, etc. The day previously he had got his feet thoroughly soaked with ice water in unloading a carload of ice. At this time he had a hard cough and Dr. Paxson found him suffering from a severe bronchitis, which was relieved by treatment. He persisted in exposing himself to the night air and indulging in stimulants and the cough returned with renewed severity, and on the evening of March 20th he had a severe hemorrhage from the lungs. Dr. Paxson thinks he lost three or four pints of blood at this time. The hemorrhage yielded with difficulty to treatment and there was still some hæmoptysis when he was admitted to the hospital the following day.

Upon admission he was very much prostrated, his pulse was feeble and very frequent. Orthopnœa.

March 22. Dr. H. C. Deaver, the resident physician, was called to his bedside at 6 A. M., and found him with another very profuse hemorrhage from the lungs. Ice bags were applied to his chest, and gallic acid, 20 grains, was administered, and ergotine hypodermically. He lost about three pints of blood. His pulse was very weak and dyspnœa very great. His temperature was 102°, pulse 140. Later in the day he was ordered turpentine gtt. xv, every four hours. He had no return

of the hemorrhage. I found both lungs anteriorly full of moist râles, but on account of his extreme weakness did not deem it wise to make a detailed examination. He had a harsh, laryngeal cough, and was not able to speak above a whisper. No improvement in his general condition occurred, his temperature was continuously high, and he died on the afternoon of the 26th of March.

The post-mortem examination revealed cheesy masses in both lungs, very little lung tissue in a condition approaching the normal remaining. In the right lung clots were found in numerous branches of the pulmonary artery. The kidneys both showed commencing fatty change and were intensely congested. The spleen showed a cheesy nodule the size of a hazel-nut. *May* 13, 1886.

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10. *Lungs and kidneys from a case of phthisis in a syphilitic child.*

Exhibited by Dr. G. E. de Schweinitz.

Emma R., æt. 6½ years, a white child, was brought to the Children's Hospital September 25, 1885. She was manifestly the subject of hereditary syphilis, and presented the old and worn look, harsh, pitted skin, depressed nose, scarred angles of the mouth, and dental malformations, more or less characteristic of this condition. The superior central incisors did not present the crescentic notch of Hutchinson, but the teeth were transversely grooved in the crown, offering a type which may be placed in the group of erosions "*en sillon*" of Fournier's classification. In addition, there were scattered over the body, chiefly on the forehead and outer aspects of the forearm and legs, large rupia-like sores, covered by heavy piled-up crusts, and leaving, after healing, pigmented scars. From the nostrils there escaped a constant sanious and irritating discharge. Long standing inflammation of the conjunctiva had produced an essential atrophy of this membrane, and a contracting scar-tissue, which bound the lids to the ball, and caused an incurvature of their ciliary borders, so that the lashes rubbed constantly against the globe of the eye and the lower margin of the cornea. After some weeks of appropriate constitutional treatment, I performed an operation (Yäsche-Arlt) for the relief of the deformity of the lid, with distinct benefit. After a time, however, the contracting tissue between the ball and the lid was reëstablished and, as it caused considerable pain to the child, another operation was deemed advisable. The ball was dissected loose,

and, with the hope of preventing reädhesion, a flap of rabbit's conjunctiva transplanted. This operation met with indifferent success, and the graft soon melted away. About this time the pulmonary symptoms became evident by a constant cough and evening rise in temperature.

Examination of the lungs showed first consolidation of the apices, and later on, in succession, the physical signs of softening and cavity formation. The nutrition of the patient steadily declined, night-sweats appeared, the temperature varied from 98.5° to 100° in the morning, and 103° to 104.5° in the evening. The course of the disease from this time on was rapid, and death took place toward the end of April of this year.

Post-mortem examination.—This I made twelve hours after death, assisted by Dr. Wharton, and Dr. Cochran, the house surgeon. Slight rigor mortis. Position of viscera in thorax and abdomen normal.

Thorax: Right lung. Dense adhesions at apex, and anteriorly and posteriorly as far as the fifth rib. In the apex there was a small cavity, or rather succession of cavities, filled with greenish-yellow pus. Below this, and anteriorly as far down as the fifth rib, an area of breaking down filled with like-colored pus. Her lower lobe was more solid, but on section pus exuded from numerous minute cavities, and exhibited in intervening tissue numberless yellowish-gray masses (tubercles). Posteriorly, in lower lobe, directly beneath plura, there was a large area of breaking-down tissue. Left lung: There was an apex cavity filled with pus, and a general condition of the lung similar to the one just described, only the process had not advanced as far as in the right side.

Heart and pericardium: Usual pericardial fluid. Firm chicken-fat clot in the right heart, and a smaller one in the left heart. Valves normal.

Abdomen: Liver about normal in size and weight, and presented no abnormalities except a depressed white scar in the right lobe.

Kidneys of natural size, capsules easily stripped off, the medulla (central part) of the right kidney occupied by a dense, hard, reddish-white mass.

The mesenteric glands were enlarged. The rest of the abdominal organs showed no gross lesions. The brain and cord were not examined.

Just what the duration of the pulmonary lesion was I cannot say, inasmuch as, unfortunately, no careful examination of the lungs was made until six or eight weeks before death; because, up to that time no clinical symptom had called attention to the chest. The lungs present the appearance of tuberculous organs, but as they occurred in a child

the subject of hereditary syphilis, a careful microscopical examination will be made, a report of which I will present at the next meeting.

May 27, 1886.

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11. *Foreign body removed from the bronchus after tracheotomy.*

Exhibited by Dr. H. R. Wharton.

A steel glass-headed pin, one and seven-eighths inches in length, which had remained impacted in the bronchial tube for twenty-three months, and was finally coughed up. Tracheotomy had been performed in this case for the marked dyspnœa which occurred a short time after the introduction of the foreign body into the air passages, which was followed by much relief as regards the symptoms of dyspnœa. The case made a perfect recovery. *June* 10, 1886.

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12. *Case of localized encysted empyema with right pleural effusion, apparently complicating typhoid fever* (?).

Exhibited by Dr. H. M. Fisher.

James H., single, æt. 20 years, a dyer by trade, was admitted to the Episcopal Hospital March 5, 1887. For the following notes I am indebted to Dr. Ziegler, the resident physician.

The patient appeared to have been in good health up to the time of present illness. Ten days before his admission to the hospital he began to suffer from headache and general malaise and diarrhœa. He was obliged in consequence of these symptoms to stop work, but did not take to his bed until one week later, when he was suddenly seized with a "stitch" in his right side, and shortness of breath. Upon admission he was drowsy and dull, and had slight muttering delirium. His face was dusky red, his hands somewhat livid, tongue dry and red, and sordes on the teeth. He had diarrhœa, the stools being grayish; his abdomen was tympanitic; there was not much apparent tenderness, but some gurgling in the right iliac fossa, and one or two faintly red colored spots. He had a good deal of cough, with scanty expectoration, and much dyspnœa. The apex-beat was detected one and a half inches to the left of the nipple, and there was abnormal resonance over the sternum. On the right side, percussion resonance was exaggerated from the clavicle as far as the

fourth rib, and below this point the note was absolutely flat. Posteriorly there was impaired resonance over the whole of the right chest, but vocal fremitus appeared not to be markedly diminished.

Crepitant râles were heard at the lower angle of the right scapula, and friction sounds were heard in the right axillary region. In this region the respiratory murmur was inaudible. Under the left clavicle moist râles were heard, and over the whole of the left lung the breathing was harsh. Some slight impairment of percussion resonance, and a few subcrepitant râles at base of left lung.

March 6. Dyspnœa has increased; bronchophony and bronchial breathing at right mid-scapular region. Other signs persist.

April 7. Friction sounds heard to-day under right clavicle.

8*th*. Weaker and more delirious. His temperature, which, during the last two days, has varied from 100°–103°, was 104° at ten o'clock this morning. Has from six to eight loose passages daily.

9*th*. Heart sounds feeble, and pulse frequent and small. Temperature at 10 A.M. 103$\frac{2}{5}$°. Much dyspnœa.

10*th*. Died at 6 A.M.

An autopsy revealed a congested condition of the mucous membrane in the lower portions of the ileum, and a swollen and injected condition of Peyer's patches, without any apparent ulceration, and the mesenteric glands were much swollen.

In the anterior mediastinum there was an encysted collection of pus, of apparently about six ounces in amount. The accumulation was cut off from the cavity of the right pleura by tough pseudo-membranous septa, and on the left side there was a band of false membranes closely adherent to the pericardium. The inner surface of the pericardium showed no sign of inflammation. No communication could be traced between the above mentioned abscess cavity and a bronchus. The right pleural cavity contained about a quart of clear serum, and over an area of about three inches square there were firm adhesions between the costal and visceral layers of the pleura. There was slight congestion of the bases of both lungs, but no consolidation. *December* 23, 1886.

13. *Two cases of very large rhinoliths.*

Exhibited by Dr. C. Seiler.

The history of rhinoliths is very meagre in the number of cases reported, and we find that Cohen, in his admirable work, dismisses the subject with a few lines. Mackenzie, of London, in his book, devotes a little more space to rhinoliths, and gives a condensed history, in which, however, but few cases, comparatively speaking, are enumerated, in none of which the stones seem to have been as large as in the two instances presented to-night. Rhinoliths are calcareous concretions, resulting from the impaction and decomposition of the natural secretions of the nasal mucous membrane, and are usually started by the impaction of foreign bodies in the nasal chambers. They are usually of small size, but by aggregation may grow, if retained long enough in the nares, to considerable size. Their close adaptation to the configuration of the inner surface of the nasal cavities makes it impossible to remove them in their entirety, and, therefore, large specimens are rare. Chemical analysis shows them to be true calcareous concretions.

The first specimen presented was sent to Dr. Formad by Dr. J. C. Gable, of York, Pa., with the following short history. Dr. Gable writes:

"On December 3, 1886, Mr. A. D., æt. 23 years presented himself in my office for nasal examination and professional advice. I obtained the following history: Patient born in Windsor, York Co., Pa., his present place of residence. He suffered during the last ten or twelve years with constant and severe headache, obstructed nasal respiration, almost complete in the right nasal cavity, nasal voice, anosmia, somewhat inspissated, slightly offensive and excessive nasal discharge, slight chronic irritation of the throat, and an appreciable amount of excessive convexity of the bony portion of the nose on the right side. Until I saw the patient he had received catarrhal interpretation as the cause of his sufferings. Upon examination I found a foreign body in the right nasal chamber, which, when viewed by reflected light through a nasal speculum, could be readily made to move by an ordinary grooved director introduced through the speculum. The mobility was, however, but slight. I introduced a common dressing forceps, and by considerable effort succeeded in crushing a small fragment, which, upon examination, I found to be that of a rhinolith.

I then, without administering an anæsthetic, attempted to extirpate the stone by crushing and remove it piecemeal, but found it so difficult

and painful to the patient that I was obliged to resort to ether, when, with the same forceps, I succeeded with considerable difficulty in crushing small fragments from the concretion, which so reduced it in size that after a number of strong tractions I succeeded in removing the main body *en masse*. Upon examination after its removal, I found considerable laceration of the interior of the nose, with some deviation of the nasal septum to the left, and an unusually large space in the nasal region between the inferior and middle turbinated bones, where the main body of the rhinolith seemed to have been located, with its thickest end pointing downward, its convex surface outward, and its plain surface toward the septum. Since the operation the patient has almost entirely recovered from the headaches, anosmia is improved, nasal respiration free and easy, and the nasal tissue has returned to an almost normal condition. The general condition of the patient has been, and continues, good."

The second specimen I removed from a patient on December 2, 1886, who related the following history:

D. S., æt. 32 years, said that he had suffered for more than twenty years from nasal obstruction, particularly on the left side, which had caused shortness of breath, headache, loss of memory, and fetid breath, particularly within the last two or three years. The odor was complained of particularly by his wife, who said that frequently in the morning it made her sick. On examination of the nose, I found the left nasal chamber occluded by a deviation of the cartilaginous septum, the edge of which came in contact with the ala. Thinking that this was sufficient cause for all the symptoms complained of, I removed the projecting portion by excising it, and then discovered that the whole of the nasal chamber was filled with a foreign body, thoroughly attached to the turbinated bones. By the rhinoscopic mirror it was seen to come within a quarter of an inch of the posterior edge of the bone. An attempt at removal with the forceps caused so much pain and hemorrhage that I decided to give the patient ether, and thoroughly cleanse the nasal chamber of the obstructing material, which, up to the time of the operation, I thought was necrosed bone, although no syphilitic history could be obtained.

The following day, December 2d, Dr. Reeves gave ether, and I began to extract the supposed necrosed bone. I very soon, however, found that I had to deal instead with a calcareous concretion which was too large, and too closely adherent to be extracted in toto, and I therefore introduced a large polypus forceps and crushed it. Many small pieces came out with the blood and were lost, so that the specimen shown represents only about one-fourth of the entire mass. A good sized mucous polypus

was extracted at the same time. Although the hemorrhage was quite profuse, and the laceration of the mucous membrane considerable, the patient made a rapid recovery, and is, at the date of writing, free from all the symptoms complained of before the operation.

January 13, 1887.

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14. *Empyema.*

Exhibited by Dr. F. P. Henry.

Joseph L., German, cigarmaker, æt. 28 years, was admitted to the male medical ward of the Episcopal Hospital on the morning of April 4th complaining of a severe pain at the base of chest and a very troublesome cough. Temperature was normal, pulse 80. No previous history could be obtained.

No physical signs on examination of chest except that there was slight impairment of resonance at the base of chest. A diagnosis of diaphragmatic pleurisy was made. ¼ gr. morphine sulph. was given every four hours until pain was relieved. No friction sound was audible. The man was relieved by the morphine and smaller doses were given at regular intervals.

April 9. Breath sounds distant and feeble at the bases of both lungs. Patient says he feels better. Cough is much better, no expectoration. The temperature in the evening was 100°.

17*th*. He complained of pain at the base of left scapula. Bronchial breathing over lower half of the chest posteriorly. Temperature ranged between 100° and 101° until the morning of the 20th, when it fell to 97°. Bronchial breathing was not so marked, and subcrepitant râles were heard over lower half of chest. Expectoration was very much diminished and the patient said he felt much better.

24*th*. On making my morning visit I found the patient looking very pale with a feeble pulse and great dyspnœa, and complaining of intense pain over left chest. On percussion the note was tympanitic, and amphoric breathing was heard all over chest; a splashing sound was very audible on shaking the patient. Stimulants and morphia were ordered. The next morning he was aspirated, when I drew off fourteen ounces of purulent liquid. This seemed to give him relief for the time; he gradually grew worse until he died.

The *autopsy* showed the left chest full of pus and the lung collapsed.

May 12, 1887.

V. GENITO-URINARY APPARATUS.

a. KIDNEYS, BLADDER, ETC.

1. *Enlarged prostate.*

Exhibited by Dr. G. HINSDALE.

The specimen had recently been presented to the Mütter Museum of the College of Physicians, by Dr. J. L. Stewart, of Erie, Pa. The specimen had been removed from a man, æt. 75 years, who, sixteen years previously had first come under Dr. Stewart's observation. At that time he was a strong, well-developed man who had never been ill in his life, but was then suffering from retention of urine which had existed for seventy-two hours. It was found to be impossible to introduce a catheter owing to an enlarged prostate, estimated to be larger than a hen's egg. Circumstances rendered it necessary to force an instrument through the gland and five and a half pounds of urine were drawn off. A train of most unpleasant symptoms followed and for weeks there was profuse suppuration with complete incontinence and great prostration. After about thirty days improvement began and continued to complete recovery. Three months afterward the patient seemed perfectly well.

Attacks of cystitis and retention became frequent, and for sixteen years only once did an interval of more than three months pass without an attack, the usual time being about twenty days. During this time Dr. Stewart introduced the catheter 1194 times. Pain was intense, during the later years, when four or five ounces of urine had collected in the bladder. Meantime the prostate continued to increase in size, and in November, 1884, was believed to be of the size of a large orange. On the night of the 27th of May, 1885, the patient had his last attack. Dr. Stewart not being at hand, two other physicians did not succeed in introducing an instrument. Just before 9 A. M. of the following day the man was attacked with the most excruciating pain followed by a severe chill. At this time it is believed by his medical attendants that rupture of the bladder occurred, and the early date of this accident is accounted for as being the result of the contracted condition of the bladder.

From this time there was no acute pain, but a severe aching followed by prostration. At 9.30 A. M. the bladder was aspirated, one ounce of urine coming away. Dr. Stewart catheterized him on the third day, drawing about a tablespoonful of urine. The patient died on the morning of the fourth day. His mind was clear and his voice strong to the last.

The *post-mortem* examination was not made by Dr. Stewart personally. It was stated that there was a rupture of the anterior part of the bladder near the fundus and that the cavity of the abdomen was filled with urine. The specimens were not removed in such a way as to make this evident.

The specimen, as presented, consisted of the prostate gland laid open by a cut in the vertical line and having attached to it the bladder, the walls of which had been cut in several directions. These walls were thick and had apparently undergone fatty degeneration, as had also the kidneys, which accompanied the specimen, the pelves of which were thickly overlaid with fat. The long diameter of the prostate, after being in alcohol for three months, was three inches; the shorter diameter, two and three-quarters inches; the third lobe was one inch long, and through it the catheter passed and still remained in position. The bladder walls when replaced indicated a very small internal capacity.

September 10, 1885.

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2. *Kidneys, ureters, and bladder from a case of death following the operation of litholapaxy.*

Exhibited by Dr. G. E. DE SCHWEINITZ.

Dr. Agnew kindly permits me to exhibit these specimens, which were removed from a patient of his in the University Hospital. The man, æt. 73 years, for two years previous to operation had suffered with straining during the act of micturition, a desire to make water with undue frequency, a feeling of weight and distention about the perineum, irritation of the rectum, and other symptoms of chronic hypertrophy of the prostate gland. Such, indeed, had been the diagnosis until Dr. Agnew examined him and found a vesical calculus. In the patient's clinical history there were no points of special interest, except that he had been a steady whiskey drinker. Examination of the urine showed sp. gr. 1018. Sediment: albumen, pus, epithelium, and granular casts.

The operation of litholapaxy was performed, marked difficulty being experienced in the introduction of the instruments. After the opera-

tion the patient exhibited great restlessness, præcordial pain, and a rapid, feeble pulse. Later he became comatose, and died twenty hours after the operation, the immediate cause of death being, apparently, the formation of a heart clot.

Post-mortem examination, ten hours after death.—Body well made, nothing of note anteriorly, the usual posterior ecchymoses.

Thorax: Old pleuritic adhesions on the right side, left side free. Lungs crepitant, with slight posterior congestion. Heart surrounded with fat, muscular fibres flabby, valves normal, firm chicken-fat clot in right ventricle.

Abdomen: Position of viscera normal, marked deposits of fat in the omentum. Liver slightly enlarged, soft, but otherwise normal. Both kidneys granular and containing cysts. Bladder thickened and showing two spots of ecchymosis on the mucous surface. Prostate enlarged, other organs normal. Brain and membranes not examined.

Dr. Tyson thought this case added one more to the list showing the impropriety of crushing for stone when kidney disease was present. He did not know why, but under such circumstances the cutting operation was more suitable. Dr. Tyson cited the case of Louis Napoleon as an instance of the danger of crushing in the presence of kidney disease. Dr. Tyson said, in answer to a question of Dr. Barton's regarding the kidneys, that they were contracted, though not markedly so, as there was good secreting structure left. The cysts were part of the pathological anatomy of granular kidney. He believed the cause of death to be uræmia.

Dr. G. G. Davis said that in such cases a post-mortem examination was in reality of the nature of an investigation. Death might be due to direct violence done to the urethra, the prostate, or the bladder; or it might be due to extension of inflammation to the peritoneum, or to an unknown cause, as in the present case.

Dr. de Schweinitz said that the urethra was not examined, and agreed with Dr. Tyson that the cause of death was uræmia. This was the more probable because an officious attendant, against orders, had administered rather full doses of morphia. *September* 24, 1885.

3. *Cystic kidney; acute inflammation of a cirrhotic kidney.*

Exhibited by Dr. J. H. Musser.

This specimen was removed from the body of a male, æt. 72 years, who died of apoplexy, in October. As can be seen, the kidney is about the normal size but quite cirrhotic, though its capsule was removed readily and its surface is smooth. The left kidney presented the same degree of cirrhosis. In this one, however, in addition, two cysts are seen. The large one occupies the upper quarter of the kidney. It was larger than a walnut and distended with clear fluid, in which floated cheesy flocculi, as observed in this bottle. The walls of the cyst are thickened and opaque in several places, due to a deposit of cheesy mucus on its inner walls. The cheesy matter, doubtless, resulted from the contents having been previously purulent. The cyst was partially subdivided by a membranous projection from the walls adjacent to the kidney.

The antecedent history of the patient from whom this specimen was removed, briefly is as follows: Occupation, watchman; habits, good; previous health good until he came under my care, in the spring of the present year. This was on account of lumbar pain and œdema of the legs and face, which developed suddenly without chill or fever. At first his urine was decidedly bloody in appearance, sp. gr. 1025—three-fourths albuminous—and contained blood and granular casts. When he went to the country, in June, his œdema had disappeared and the urine was clear in color, normal in specific gravity, very faintly albuminous, and contained a few hyaline and granular casts. The lumbar pain continued more or less, and during the summer, I am informed, was unusually severe in the right renal region. Was the pain due to the large cyst? There was never any evidence of renal calculi.

The bloody urine, the pain, and the marked depression continuing about six weeks without amelioration, led me to fear the development of renal carcinoma, although there were no physical signs of the disease My fears proved groundless, but were intensified by the fact that at the time I was attending a sister, who since died of carcinoma. I call this an acute inflammation of a cirrhotic kidney, for it seems to me the appearances and the cyst indicate a chronic process of a longer duration than the symptoms would indicate. *November* 12, 1885.

4. *Diabetes mellitus; diabetic coma; absence of fat embolism; vesical calculus.*

Exhibited by Dr. J. H. Musser.

The patient, after a journey of two hours in a rough-riding wagon, was admitted to the Presbyterian Hospital, November 7, 1883, whither she had come to be operated on by Dr. Girvin, for the relief of a vesico-vaginal fistula. She was æt. 43 years, of good habits, and of robust appearance, and good health until recently. The fistula had been acquired in 1871.

Two months before admission she noticed increased thirst and more abundant urinary secretion. Debility, but not emaciation ensued, so that three weeks ago she had to remain in bed most of the time. On admission the above symptoms were noted. She was unusually fat; she had no appetite and was constipated; her vision was not disturbed, and there were no cerebral symptoms. The urine was clear, sp. gr. 1026, non-albuminous, but largely saturated with sugar.

The resident physician, Dr. Hodges, observed that she was much more drowsy than natural and quite dull of intellect the third and fourth days after entrance, and on the fifth she was semi-comatose with rapid pulse and hurried breathing. The morning of that day she complained of epigastric pain. At 1.30 P. M., her temperature was $102\frac{2}{5}°$. At 5 P. M., she was comatose; respiration 64, labored; temperature not much increased to the touch. Death at 7 P. M.

Autopsy, three hours after death.—Rigor mortis slight, in legs only. Ecchymoses on posterior surface of legs and trunk. Body well nourished and warm. Temperature in the abdominal cavity $107\frac{4}{5}°$; in mouth 98°.

Brain not examined.

Thorax: Pericardium normal. Left ventricle of heart contracted; slightly hypertrophied; all valves normal; no clots, dark blood in cavities. Lungs contained air. No hypostasis or œdema. Aorta slightly atheromatous.

Abdominal cavity: Omentum loaded with fat. Peritoneum (parietal) thickened and congested. Stomach normal. Pancreas normal. Splenic capsule shrivelled, tufts prominent, size normal. Liver very large, fatty, and congested. Gall-bladder partially distended with bile and contained thirty stones. Kidneys enlarged and fatty. Uterus normal; bladder walls thickened; fistula between bladder and vagina; in bladder a large phosphatic calculus.

The muscles of the body were intensely red. On opening the body a decided acetous odor was observed, which was much more apparent when examining the blood. The blood removed from the vena cava had a most marked lactescent appearance, and on standing twelve hours, while not coagulating, globules of fat, distinct after immediate removal, were very readily seen. The lacteals in the mesentery were engorged with chyle. The blood and urine were not tested chemically; the latter could not be collected.

Microscopical examination of the liver showed it to be highly fatty, with a slight overgrowth of connective tissue, but no so-called hypertrophic cirrhosis. The lung sections, stained with osmic acid, did not show any evidence of fat emboli. The kidneys were fatty and slightly cirrhotic. The pancreas was normal.

The important facts in this case seem to be the short duration of the disease; the development of coma after fatigue; the high temperature with a possible post-mortem rise (the temperature was taken on account of the extreme heat of the interior of the body); the fatty blood without fat embolism; the coexistence of a vesical calculus, which, however, developed prior to the diabetes, and, no doubt, on account of the bladder injury. *November* 12, 1885.

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5. *Congenitally misplaced kidney.*

Exhibited by Dr. J. B. Deaver.

The kidney was taken from a body in the dissecting-rooms of the University of Pennsylvania. It was the right, and was situated opposite the body of the fourth lumbar vertebra, resting upon the aorta and common iliac arteries and veins. It derived its blood supply from the aorta and common iliac by two arteries. The two veins emptied into the ascending vena cava and common iliac. The ureter sprang from the anterior surface and entered the bladder normally. There was a complete absence of a mesonephron. *December* 10, 1885.

6. *Case of encephaloid sarcoma of kidney, and probable dissemination through traumatism.*

Exhibited by Dr. I. P. Stritmatter.

Theresa M., æt. 5 years and 1 month, came under my care July 27, 1887, with the following history: Father and mother living and well. Of two paternal aunts, one died of phthisis at forty; the other living and well. Maternal grandmother died of some disease accompanied by ascites, for which she was tapped twelve times during a period of fifteen months. Of seven maternal uncles, one died of Bright's disease; others and one maternal aunt living and well. Hence, no history of malignant or other growth. I first saw this child June 29th, while attending her father. I was told she complained of pain in the left side of the abdomen, and would say she had worms. Being in a hurry, I made only a superficial examination. It was sufficient, however, to convince me of the existence of some hard body. At my next and last visit to the father I did not see the child. She was brought to my office on July 27th, when the growth in the left side could be made out distinctly. The mother then told me that on July 4th this year, the child fell from the truck of a wagon on the brake-handle, a distance of not over one and one-half feet, striking her left side. She did not complain more than usual of pain after a day or two, and when brought to me it was not that she suffered, but because of her protuberant abdomen. The tumor was then made out as extending to and under the ribs of the left side, a nodule to the median line on a level with the umbilicus, and a second nodule a little lower, extending beyond the median line. Tumor was not tender to touch; it could not be moved easily and was non-fluctuating. Child in pretty fair health.

From this date to September 17th, the child began to fail, and the tumor increased in size rapidly. September 15th, the child's abdomen began to be tympanitic and tender to the touch. Temperature varied from subnormal to 99–100°. She was sent to Dr. Montgomery, and after examination under ether it was decided not to operate. Since then child had had little pain. October 15th the feet became œdematous, and later the nates and vulva. Coincident with the œdema, two veins became very prominent; one on left extending to the lower ribs; one on right into the right axilla. During the last week there was a prolapsed and œdematous condition of the bowel. During the whole time there was normal or subnormal temperature, with the exception of one week, when crackling over abdomen with rise of temperature occurred. Died November 26th, 5 A.M.

Post-mortem examination, thirty-one hours after death.—Drs. Montgomery and Waugh present. Body extremely emaciated, axillary and abdominal veins enlarged. Lower extremities, nates, and vulva œdematous, and bowel everted. Incision revealed mass of round white bodies studding omentum and completely enveloping bowels. Seemed to have principal attachment in region of kidney and bodies of vertebræ.

Right kidney enlarged to size of fœtal head and sarcomatous; no kidney structure visible. *Left kidney* normal. *Liver* studded with nodules which broke down on pressure. In its great fissure was a growth resembling a bunch of grapes; a like mass on upper surface between liver and diaphragm. *Spleen* imbedded in same mass with left kidney, also nodular. *Right lung* had two nodules in apex; the rest, and the *left lung* and *heart*, unaffected.

Brain and *cord* were not examined.

In reviewing this case, there are several points of peculiar interest. In the first place, the rapidity of growth and the probable influence of traumatism in its dissemination. There are a number of cases reported of similar growths in the kidney occurring in children of same age, and, I believe, Dr. Formad has such a specimen before the Society this evening, taken from a child two years of age. But in all these cases I can find none in which the abdominal organs, and particularly the omentum, are involved.

A study of this case has led me to the belief that this growth had been in existence for a considerable time, limited to the right kidney, and that to the fall on July 4th are mainly due its rapid growth and dissemination.

Dr. Formad, to whom I sent a specimen for examination, will kindly furnish the Society with a report of histological character of growth.

Dr. Formad said that he had examined the growth and found it to be a congenital rhabdo-myoma, which had undergone sarcomatous change. Primary renal sarcomata rarely give rise to metastasis, and he accounted for the numerous secondary growths in this case by the fact that the sarcomatous change was secondary to the rhabdo-myoma.

December 10, 1885.

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7. *Cystic kidney, removed by operation.*

Exhibited by Dr. C. M. Wilson.

The specimen presented to-night was removed five days ago by abdominal section, from Johanna K., æt. 42 years. The patient first

came under observation three weeks ago, at which time she was suffering from an attack of renal colic, accompanied by hæmaturia. The specimen is that of a large cystic kidney, and one that has probably undergone sarcomatous degeneration. I regret to say that no microscopic examination was made. Decomposition occurred so rapidly that this was not possible. The specimen measures eight and a quarter inches in its greatest length, and four and a half inches in its greatest width; its weight is eighteen ounces. Its peripheral surface is studded with small cysts. The pelvis was as large as a goose's egg, and filled with pus and recent coagula. Drs. Osler and Formad think it a cystic kidney, and do not believe that malignant disease is present—a point only to be determined by microscopic investigation. The patient lived 105 hours after the operation, and died of uræmia. *February* 25, 1886.

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8. *Case of interstitial nephritis.*

Exhibited by Dr. H. M. Fisher.

Was called January 19, 1886, to attend E. A. B., colored, æt. 76 years, widow. Her general health had been fairly good for a woman of her age, except a tendency to rheumatic pains, and stiffness in her joints. She had been complaining since early morning of abdominal pain; she had also had nausea and vomiting. I found her with rather a feeble pulse (92), and suffering from dyspnœa (respiration 44). Though she complained of a great deal of pain in the upper part of the abdomen, there was not any very marked tenderness. Tongue much coated. Ordered a purgative (saline). The following day, no movement of the bowels having occurred, the purgative was repeated. Examination of the heart showed moderate hypertrophy, but no murmur was detected. Examination of the lungs showed everywhere hyper-resonance and feeble breathing, but no râles were heard. Urine, specific gravity 1022, very cloudy; urates in large excess, and a trace of albumen. Microscope showed in sediment urates, pus corpuscles, bladder epithelium, and a very few *hyaline casts.*

January 21. Bowels still sluggish. Ordered hydrarg. chlorid. mitis, gr. v; sodii bicarb., gr. x; to be followed, if necessary, in eight hours, by half an ounce of castor oil. Pain undiminished. Ordered also tincture of digitalis, fifteen to twenty drops, every four hours.

22*d*. Still no movement of the bowels. Ordered an injection. This had the desired effect, bringing away copious discharges. Patient expressed herself somewhat relieved after this, but toward evening her pulse got very weak, and she died at midnight.

The *post-mortem examination* was made about fifteen hours later. I was informed by the undertaker that there had been a good deal of post-mortem purging. Very thick panniculus adiposus.

Lungs everywhere highly emphysematous.

Heart: Left ventricle rigidly contracted and empty; right ventricle dilated. Valves all apparently normal. Weight of heart twelve ounces. Much fat on surface of organ.

Kidneys: Left kidney weighed three and a quarter ounces; right kidney four ounces. Both of them were *granular* on section, and showed *cortex reduced* to one-eighth or one-quarter of an inch. Capsules somewhat adherent. Cysts on upper convex surface of right kidney.

Small intestine, from a point about a foot from the pyloric orifice, showed on its outer surface a livid redness. This appearance was seen over fully two yards of the intestine. Internally, this redness appeared to be due to stasis of blood in small veins. No ulceration of mucous membrane anywhere apparent.

The *liver* was rather firm in texture, and showed catarrhal inflammation of biliary ducts. *May* 13, 1886.

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9. *Dilatation of ureter and hypertrophy of the bladder.*

Exhibited by Dr. J. Daland.

M. S., æt. 9 years, female, white. In 1880 had scarlatina, since which time she has complained of polyuria, frequent micturition, thirst, gradual loss of color and strength, slight irregular fever, rapid pulse. Usually passed four to six pints of urine daily, and examination of that secretion showed a moderate amount of albumen, no sugar, moderate amount of pus, no blood or tube casts. There was a temporary improvement and then she became feverish, pulse rapid, vomiting. Remarkable over-action of the heart. She gradually grew worse and died.

Autopsy, two days after death.—Kidneys small and shrunken. Left kidney measured two inches in length, and one and three-quarters inches in width. The calices, infundibula, pelves, and ureters were all enormously dilated. In places the ureters measured one inch in diameter. The walls were remarkably thickened, chiefly due to hypertrophy of the

muscular coat. At the beginning, and just before they terminated in the bladder, the lumen of the ureters suddenly diminished, though still considerably larger than normal. The mucous membrane of the whole urinary tract seemed thickened. The vesical mucous membrane was covered all over with small grayish spots. These pigmentations may have been due to old ecchymoses.

The bladder was greatly enlarged and its walls thickened. Here, too, the muscular coat seemed the chief cause of the thick walls, which, in places measured two to three lines. The capacity of the bladder had increased fully threefold. The mucous membrane seemed slightly thickened. Urethra normal. There was no calculus and no history at any time of urinary obstruction. The sequence of events seems to me to have been somewhat as follows; After the scarlatina in 1880, excessive urination occurred and this persisting necessitated an increase in the capacity and thickness of the walls of the bladder, and consecutively the same condition in both ureters and pelves. Frequently she would void more than a pint of urine at one time. The heart showed simple hypertrophy. The remaining organs were normal. The brain and spinal cord were not examined. *May* 27, 1886.

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10. *Contracted bladder, dilated ureters, and surgical kidneys from stricture of the urethra.*

Exhibited by Dr. T. R. Neilson.

The patient, from whom this specimen was obtained, came under my care about fifteen months before his death. He dated the beginning of his trouble about a year earlier than that, complaining chiefly of frequent calls to pass his urine, especially at nights. Exactly one year before I saw him, but some months after he first noticed trouble with his urinary organs, while climbing a tree he slipped and fell about ten feet, striking the inner aspect of his left thigh just above the knee upon a branch of the tree. He stated positively that there was no visible injury to the perineum or external genitals, nor to his back. For the next three or four months, he passed a few drops of blood at the end of every act of micturition; he placed himself under the care of a physician in the country, where he was working, and before long the bleeding ceased.

At the time he placed himself under my care I made the following note of his condition: His age was 36 years, he was married, had never had gonorrhœa. He was compelled to pass water about eighteen times in the twenty-four hours, oftener at night than during the day; he was thin, looked sallow, suffered from flatulence, poor appetite, low spirits, and constipation; he complained of scalding on micturition, felt at the middle of the spongy or penile portion of the urethra, the stream was twisted, and after micturition a few drops of urine dribbled from the meatus; he had a slight gleet; his urine was normal in color, but on standing a flocculent sediment was noted; circumference of penis 3 inches, strictures of 20 calibre, at ¼ inch, 2 inches, 4½ inches, and 5½ inches, and, between and behind the last two a granular condition of the urethra. I placed him upon the "gradual dilatation" treatment, being contented with a maximum of 24 calibre, on account of the grave condition that I suspected to be the cause of his symptoms. I also placed him upon such medicinal treatment as I thought suited to his general and local condition. For a time he improved, but soon became depressed in spirits, and suffered at intervals for the next month with chilliness and slight rises in temperature. At that time his urine, which was frequently throughout his case examined by my friend, Dr. Simes, as well as myself, contained a large quantity of pus. I made an examination of the condition of the prostate, with my finger in the rectum, and found it *hard*, *very tender*, and considerably *enlarged* on the *left* side, while on the *right* it was *hard* and *tender*, but *not so large*, and the *middle lobe* was *distinctly enlarged*. This latter condition explained a symptom of which he then began to complain, viz., a desire to pass more urine after he thought the act of micturition was complete, or, in other words, to rid his bladder of what Sir Henry Thompson calls the "residual urine."

Without going further into detail, I would say that the case progressed steadily toward a fatal conclusion, there being occasional periods when treatment, especially washing of the bladder, after Sir Henry Thompson's plan, afforded decided relief, and other periods when nothing that could be done was of any avail in controlling the frequent micturition, the pain in the loins (worst in the right), the occasional ill-defined chilliness and fever, and the very visible decline in the patient's strength. To afford the bladder a period of rest from the incessant straining to empty itself, I thought at one time of performing perineal cystotomy, but upon consultation with Dr. Simes, when a thorough examination was made, it was decided not to interfere. The patient passed from my care into that of a homœopath, by the well-meant, but indiscreet advice

of a lady parish-visitor, about a month before he died, but about twenty-four hours before death I was again given charge, and found him comatose and exhausted, after several days' severe suffering, and in that condition he died.

In the *post-mortem examination* I again was assisted by Dr. Simes. In the specimen, consisting of the prostate, the bladder, ureters, and kidneys, removed without separation from each other, we find a state of affairs which answers in all details to the symptoms presented during life.

The *prostate* is found to contain in its left lobe an abscess cavity as large as a cherry, its middle and right lobes being harder than normal, and the former enlarged.

The *bladder* so small as to contain barely a fluid-ounce, the quantity which was usually passed at a time toward the end of the patient's life. The walls are thickened, the mucous coat is ribbed, and in the *bas fond*, when the specimen was fresh, was a patch or region where there was still active chronic catarrh.

Ureters and kidneys: The *left ureter* is much dilated, the calibre fully 30 of the French scale, its walls are thin and flabby; *pelvis* of kidney dilated. In the *left kidney* numerous small cysts or abscesses are observable; the dimensions are—length, 4½ inches; thickness, 2 inches; breadth, 2 inches. The *right ureter* is thickened and smaller in calibre than normal for a short distance above the bladder orifice, then it becomes dilated and as large as the left one. The *right kidney* is a fine specimen of suppurative pyelo-nephritis; its length is 7½ inches, its breadth is 3 inches, its thickness about the same; the surface shows a number of large and many smaller cysts or abscesses.

Remarks.—This case is one of those which call our attention forcibly to the grave sequences that may arise from urethral stricture—especially when it encroaches so little on the calibre of the canal as not to interfere with the mere passage of the urine. Chronic prostatitis follows as one of the first evil results of the coarctation, then vesical catarrh of more or less severe type, and this once established opens the pathway, literally, for disease to reach the ureters and then the kidneys. In the instance before you, such doubtless was the course. *June* 10, 1886.

11. *Uric acid calculus of the kidney.*

Exhibited by Dr. C. M. Wilson.

This date-shaped stone was passed by a woman æt. 45 years, after seventy-two hours of intense suffering. Its weight is eighty-one grains. It is about one inch in length. A careful microscopic examination made by Dr. Judson Daland showed it to be composed entirely of uric acid. The microscope also showed the characteristic crystals; nitric acid dissolved the crystals. Burning of the fragments gave the characteristic fumes. Fracture showed that the stone was crystalline. No definite nucleus was observed. The shape of the calculus is peculiar, like a date-seed. The case presented evidence of hydronephrosis. After the discharge of the stone, which was accompanied by excessive pain, there was an increased flow of urine. The case presents but few points of interest; it is a uric acid calculus of rather more than the ordinary size.

December 9, 1886.

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12. *Congenital cystic kidney.*

Exhibited by Dr. J. P. C. Griffith.

Katie H., white, single, æt. 40 years, domestic, was admitted to the University Hospital November 7, 1886. She was at the time in such a stupid condition that no satisfactory history could be elicited. What could be learned was as follows: Her family history was good; she herself had had no serious illness until April of the present year, when she suffered with what seemed to be tertian ague, which continued for months, but from which she recovered and returned to work. About the 1st of October she suffered from a painful swelling of the right ankle which was relieved by treatment. But her general health began to deteriorate, and finally, on November 4th, she was obliged to go to bed, complaining of dull headache, nausea, and great abdominal pain and tenderness, chilliness, constipation. I have learned further from Dr. Hughes, who attended her previous to admission, that she was taken ill with all the symptoms of perityphlitis. On admission she was dull and drowsy, her temperature was 99°, pulse 96, respiration 22. Her skin was pale, eyelids slightly swollen, limbs wasted.

Physical examination of the thorax showed it to be very long and narrow with a prominent sternum. The percussion resonance of the lungs was slightly tympanitic. The heart's area of dulness was slightly increased. A faint systolic murmur was audible over the apex. The liver dulness began at the sixth rib. Upon the right side of the abdomen there was visible a slight prominence caused by some body beneath. The edge of this mass could be seen distinctly two finger's-breadth below the transverse umbilical line. During respiration it was seen to move very plainly and was supposed to be the liver. The mass could be distinctly felt; was firm, inelastic, smooth, and painful on pressure; the lower margin could not be even outlined. It continued into the right flank and reached here the crest of the ilium, and was exquisitely tender in this region; dulness on percussion existed over this area and extended to the spinal column posteriorly. The examination of the urine showed that it was acid and contained numerous leucocytes. Small amount of albumen; no casts; sp. gr. 1026.

The condition of the patient steadily grew worse. The temperature was irregular. There was a rigor on the 8th.

10*th*. The tumor in the iliac region had become more pronounced and it was evident that there were two bodies there. First, the liver, which was very much displaced downward by the peculiar shape of the thorax. Second, I came upon a mass below this and extending into the flanks. It was now larger and distinctly fluctuating. The puncture with a hypodermic needle about one inch anterior to the maxillary line and two inches above the crest of the ilium revealed the presence of pus. The diagnosis of an abscess connected with the kidney, had already been made.

No improvement in the condition of the patient occurred and she died on November 11th. On the day of her death there was a copious movement of the bowels with some blood.

Autopsy, eight hours after death.—The lungs were found to be slightly emphysematous and the left ventricle decidedly hypertrophied. The right lobe of the liver projected four inches below the seventh costal cartilage, and the left lobe an equal distance below the ensiform cartilage. Below the edge of the right lobe was seen a series of cysts not adhering to the liver, but intimately attached to the cæcum and to the first portion of the colon. Over the surface of the liver were many cysts, in size from that of a pea to a cherry; twenty to thirty were to be seen over the right lobe. The lateral margin of the left lobe was occupied by two large cysts the size of a hen's egg, filled with clear serous fluid. Further dissection showed that the cysts below the liver belonged to the right

kidney. The kidneys seemed to be completely cystic, the cysts being of the size of marbles or larger and filled with a clear serous fluid having a urinous odor and containing albumen. Beneath the organ the thickened iliac fascia was seen, and fluctuation could be felt below it. There appeared, indeed, to be an indurated mass between the space of the ilium and the psoas muscle; this tilted the lower end of the kidney upward, and this again pressed upon the liver and caused its lower edge to be so visible on respiration during life. On removing the kidney a large abscess cavity was opened, containing about a pint of pus. It had undermined the quadratus and the upper part of the iliacus. There was no erosion of the ilium or of the vertebræ. Section of the lower superficial cysts showed them to be full of pus. Two of the larger ones communicated directly with the abscess cavity. The lower and deeper cysts were free. The left kidney was also uniformly cystic; upon cutting deeply into it some ounces of pus were discharged. The ureters were thickened but pervious. The bladder normal. The lower end of the ilium was congested and Peyer's glands swollen. There was a most intense colitis. The entire colon was deeply congested and covered with grayish exudation. The mucous membrane was hemorrhagic in spots. The cæcum was black. *December* 9, 1886.

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13. *Chronic interstitial nephritis, with enormous dilatation of the right pelvis, without apparent cause.*

Exhibited by Dr. J. Daland.

A. H., merchant, white, æt. 55, married. For some years past he had intermittent albuminuria and occasional hyaline tube-casts. He emaciated rapidly, and soon became markedly anæmic. After ceasing work and carrying out a very careful plan of treatment he improved rapidly, and in eighteen months was in excellent health, though the urinary condition still persisted. Feeling so well, he returned to active business life, and was soon overworking himself as he had done before. Soon it was noticed that his strength, flesh, and color began to fail, and this condition progressed, with slight fluctuation, until his death. This case was not under my observation during life, hence this imperfect report.

Autopsy, twenty-eight hours after death.—Body emaciated; rigor mortis moderate.

Lungs highly œdematous and congested; bronchial mucous membrane in a condition of chronic catarrhal inflammation. Slight old pleuritic adhesions on both sides. No pleural effusion.

Heart normal in size, rather pale in color; consistency normal. The anterior mitral leaflet was rather thickened and shortened, the remainer of the valve normal. No pericardial effusion.

Liver pale in color, but normal in size and consistency. The hepatic, cystic, and common bile-ducts pervious.

Spleen and *pancreas* normal.

Stomach showed marked evidence of chronic catarrhal gastritis. The small and large intestine, including the appendix vermiformis, normal. The transverse colon was filled with soft feces which, during life, were mistaken for a cancerous tumor.

Kidneys: The left kidney was elongated and narrow, measuring four and a half inches in length and two inches in breadth. In places the capsule was adherent; cortex rather thin; consistency increased. In certain portions of the kidney the changes due to chronic interstitial nephritis were more marked than in others; this was particularly true in the superior third of the kidney. The pelvis of the kidney was enormously dilated, but there was no obstruction in either ureter, bladder, or urethra. No calculi were found, and the mucous membrane was normal. The right kidney was in the same condition as the left, but to a less degree. The right pelvis and ureter normal. No retention cysts. The suprarenal bodies and abdominal glands were normal. The brain and spinal cord not examined. *January* 27, 1887.

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14. *Cystic and renal calculi.*

Exhibited by Dr. T. D. Dunn.

Autopsy of Dr. G. M., male, fifty-two hours after death.

The body was greatly emaciated; some post-mortem discoloration of abdomen. Brain and chest not examined.

The stomach, liver, and intestines healthy; spleen slightly enlarged.

The kidneys were enlarged and congested. The capsules were adherent, and on their removal portions of the kidney surfaces were torn off. At the places where they were more easily separated a dark granular surface remained. In both the cortical substance was somewhat increased, in which there were numerous cysts varying in size from a pea to that of a good-sized hickory-nut. There was also a number of cicatrices, representing collapsed cysts, to which the capsules were quite firmly adherent. The pelvis of the right kidney was distended with pus and

urine, and its ureter was occluded about eight inches from its origin by a calculus.

The bladder contained about two ounces of highly colored ammoniacal urine. The walls were somewhat thickened, corrugated, and congested, particularly in the prostatic portion. It contained a uric acid calculus which weighed eighty grains. This calculus was oval; surface grayish-white, covered by minute rounded granular elevations, which the microscope showed to be uric acid crystals and amorphous urate of ammonium. There was also in the bladder a small, irregular, dark concretion about the size of a buckshot.

The vesical end of the urethra was enlarged and its mucous membrane pouting. The prostate was enlarged, but not symmetrically, the right lobe being principally involved. As a consequence, the calibre of the prostatic portion of the urethra was increased.

It is my impression that the stone in the bladder antedated the enlargement of the prostate, from the fact that vesical trouble began ten years before the period that senile hypertrophy usually begins, and that it was the cause, directly or indirectly, of the morbid changes I have described. He died worn out by suffering and the progress of the renal disease. He also suffered a great deal from digestive disturbances, which were, doubtless, reflex or due to the debility attending the vesical trouble.

February 10, 1887.

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15. *Cirrhosis of kidneys, liver, and lung.*

Exhibited by Dr. E. T. Bruen.

The specimens herewith presented were taken from the body of John W., æt. 60 years, February 11, 1887. The subject had been an inmate of the Philadelphia Hospital for several months previous to his death. He had been admitted on account of dyspepsia, with headache, and other general symptoms of weakness and debility, but at no time did he complain of cough. The immediate cause of death was uræmia.

The kidneys were small, weighing two and a half ounces each, highly granular, with adherent capsules. The liver presented the typical hob-nailed appearance; it weighed three pounds. The left ventricle of the heart was markedly hypertrophied, the valves of the mitral orifice and aorta normal, but the aorta itself was studded with patches of atheroma. The right ventricle was somewhat dilated, but no marked changes were

observed. The left lung and the lower lobes of the right lung were normal, but the upper lobe of the right lung was firmly bound to the parietes by dense adhesions between the visceral and the costal pleuræ. The entire lobe was penetrated by dense bands of fibroid tissue. Near the entrance of the bronchial tubes several cavities were found which seemed to be dilatations of the bronchi situated there. The vesicular structure of the lung was destroyed, the entire lobe being nothing more nor less than a mass of connective tissue structure.

The specimens have been presented because of the interest which belongs to such extensive cirrhotic changes, and in so many disassociated organs. It would appear that many of the changes may have sprung from the combined effects of specific disease and alcoholism. Particular attention should be paid to the pulmonary changes, because it would seem that the pleural process was primary as related to the pulmonary changes. The close relation of the pulmonary lymphatic and connective tissue structures with similar structures in the pleura, indicate that similar processes to those in question may readily originate in the lung and proceed outward to the pleura, or the reverse may be true. The remarkable localization of the process in the upper lobe of the right lung would seem to render the latter conclusion most plausible in this instance.

Should this explanation of the lesions be granted, the specimens would seem to teach that similar local connective tissue formations may follow pleurisies which occur in patients suffering from syphilis, and thus some of the cases of pulmonary syphilis can be explained. That is to say, local connective tissue proliferation can occur in the lung independently of tubercular infection, although structurally there may be no characteristic features by which the specific nature of the changes can be identified. In a similar manner the lesions in cases of phthisis attributable to the inhalation of irritant inorganic substances may be explained without recourse to a tubercular theory. *February* 24, 1887.

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16. *Large encysted vesical calculus removed through the perineum.*

Exhibited by Dr. J. M. Barton.

William M., æt. 65 years, a patient of Dr. Lambert Ott, of this city, had suffered for several years from an irritable bladder, and on one or more occasions from retention. While relieving this retention with a soft catheter, Dr. Ott detected a concretion, but was unable to find it when he introduced a solid instrument.

This experience was repeated when I saw the case with Dr. Ott on two different occasions; with varying quantities of water in the bladder, with the patient in all imaginable positions, and with solid instruments of all varieties of curve, shape, and size, we were still unable to find the calculus, though we discovered the bladder to be roughened and sacculated.

In February, with the assistance of Drs. Ott, Porter, Bissey, and others, I made a median incision through the perineum, introducing the finger into the bladder. The calculus could not be felt until a straight sound was introduced beside the finger, when the characteristic "click" was elicited from a concretion several inches beyond the tip of the finger.

After increasing the incision, making a "left lateral" of it, and incising the prostate on the same side, a portion of the calculus was grasped with a long pair of small forceps and brought within reach, but we could not extract it, as it slipped from the forceps when much traction was made. After incising the right lobe of the prostate and the superficial parts, it was discovered that the calculus was surrounded and nearly covered by mucous membrane, which was firmly attached to it. After this was separated by the finger-nail, the calculus was readily removed. It is phosphatic on its surface and weighed, immediately after removal, 660 grains.

The patient made an uninterrupted recovery, the urine ceasing to pass by the wound on the twenty-sixth day. I was prepared to make the suprapubic incision, but the field of operation was so thoroughly exposed by the opening through the perineum, that the upper opening was unnecessary. *May* 26, 1887.

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17. *Prostatic calculi.*

Exhibited by Dr. H. R. Wharton.

Two prostatic calculi, whose aggregate weight was 313¾ grains, were removed by Professor Ashhurst from a patient in the University Hospital. The patient from whom these calculi were removed had had gonorrhœa some years before, which was followed by stricture, and about five years before he noticed that the stream was diminishing much in size. Four years ago he suffered from perineal abscesses, which left him with numerous sinuses, through which the bulk of the urine was voided,

and it was for the treatment of the stricture and perineal sinuses that he was admitted to the hospital.

It was noticed, on rectal examination before the operation, that the prostate was enlarged and felt very hard.

As no instrument could be introduced into the bladder on account of the tightness of the stricture, the perineum was incised in the median line, the finger being introduced in the rectum, and resting upon the apex of the prostate as a guide. When the prostate was reached the calculi were exposed and removed, the bladder was then explored and found free from stone, and in a healthy condition.

Dr. Wharton also presented two small prostatic calculi and a vesica calculus, which he had removed from a boy about 10 years of age, by the operation of left lateral lithotomy. In this case the presence of the prostatic calculi was not suspected, and they were discovered and removed when the prostatic portion of the urethra was opened in the course of the operation.

Prostatic calculi may develop in various ways: in some cases they have their origin in the inspissated secretions of the prostatic follicles, which are believed to irritate the mucous membrane, causing phosphatic depositions, which become encrusted upon the organic matter, thus giving rise to true prostatic calculi. These true prostatic calculi, if small, are largely made up of inorganic matter; if large, it is generally found that the organic nucleus is surrounded by a large amount of phosphate of lime.

Prostatic calculi may also develop from a nucleus from the kidney, which has passed through the bladder and been arrested in the prostate. They may also arise from an abscess in the prostate, which leaves a pouch in which urine accumulates and decomposes, giving rise to a calculus; or they may also arise from a fragment of a calculus broken off and impacted in the prostate during the operation of lithotrity or lithotomy.

A prolongation from a vesical calculus may extend into the prostate, giving rise to a vesico-prostatic calculus; an example of this form of calculus was well presented in a patient whom Professor Ashhurst cut a few years ago in the University Hospital, and the celebrated case of Mr. Poland's is also an example of this variety of calculus.

It also sometimes occurs that the calculus develops in the prostate, and sends a prolongation into the bladder, giving rise to what is known as a prostatico-vesical calculus. *April* 9, 1887.

b. FEMALE ORGANS OF GENERATION.

18. *Sarcoma of uterus.*

Exhibited by Dr. H. BEATES, JR.

Mrs. P., German, matured during her sixteenth year. Throughout her catamenial life she was free from pelvic disease, and had always enjoyed excellent health. She had borne four children, and suffered no miscarriage or difficult accouchements. There was no history of heredity of neoplastic disease. Menopause at the age of 48 years. Five years afterward, while in apparently perfect health, uterine hemorrhage occurred, and reappeared with regularity. It continued five or six days, disappeared, and returned in several weeks, thus closely simulating the phenomena of menstruation. After two days, inter-hemorrhagic discharge of sanguino-purulent character occurred coincidently with pain. The further course of the disease was that of a steadily progressing asthenia. Death occurred nearly seven years after the initial symptoms.

The autopsy showed metastatic depositions in the apices of the lungs, and along their lower borders. Secondary masses, similar in appearance to those in the lungs, existed in the right and left lobes of the liver. The body of the uterus and the upper part of the vagina were involved in a new growth, with ulcerating surface. Microscopic examination showed the growth to be a small round-celled sarcoma.

January 28, 1886.

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19. *Multilocular ovarian cyst removed by operation.*

Exhibited by Dr. C. B. NANCREDE.

Mrs. S., æt. 47 years, first noticed, about seven months before operation, a tumor about the size of an orange, situated in the left iliac fossa. She had always been a healthy woman, had never had but one easy labor, seventeen years before, had never miscarried, and had been regular up to two years ago, when the menopause took place. About three weeks before first consulting me (January 6, 1886) she had been suddenly taken with a profuse flooding from the genitalia. The rest of her clinical history embraced nothing beyond repeated irregular abdominal pains, marked costiveness, and swelling of the left leg. Her pain

and suffering were such that she was urgent for relief. Vaginal examination revealed the uterus pushed forward and upward, and so flexed and twisted as to defy the introduction of a sound. There was apparently a hard, immovable mass, filling up somewhat more than the whole left side of the true pelvis. This did not fluctuate, while the remainder of the tumor did. The diagnosis of a multilocular ovarian cyst, firmly adherent to the true pelvis, with practically no pedicle, was made. Again, the same almost complete absence of pedicle, it was thought, might account for the apparent fixity of the tumor, although it was feared that both conditions obtained. Assisted by Drs. Joseph Price and H. A. Kelly, on January 17th, in the presence of the surgical class of the Polyclinic School, I proceeded to remove the growth. An incision of about eight inches from the umbilicus to just above the pubes was found necessary. One adhesion each to the omentum and large bowel was tied and divided, the cyst was tapped, and the tumor was then seen apparently fusing with the uterus and the left side of the brim of the true pelvis. Doubtless this intimate connection with the uterus accounted for the hemorrhage, supposing the oviduct to have remained patulous, since one of the cysts contained a large amount of bloody fluid. After a vain endeavor to separate the pelvic adhesions a very strong double ligature was made to embrace by transfixion the left cornu of the uterus, and what remained of the broad ligament in the sulcus between the uterus and tumor; this was continued some distance down, as the "cobbler's stitch," and the tumor cut partly away. Next, by a series of partial enucleations, clampings with Wells's forceps, and transfixings of the bases of the adhesions by an aneurism needle threaded with a double ligature, and tying with the Emmet knot, after three hours of the hardest labor I have ever undergone, by the able assistance of Drs. Price and Kelly, the whole tumor was removed. Somewhat over forty ligatures must have been used. The uterine portion of the pedicle —if it may be so termed—was touched by the thermocautery, as well as one or two other points, and a few more ligatures were applied to bleeding points deep down in the cavity of the pelvis. A glass drainage tube was introduced, and the wound closed with prepared silkworm gut. Iodoform was dressed along the line of the wound, the tube plugged with absorbent cotton, dressed with the same, and the dressing was completed by a large pad of cotton and a binder pinned over all. The patient, after operation, had a pulse under 80, and recovered well, with the exception of a sinus left by the tube, which is only now slowly closing. The tumor was about the size of the uterus at term. It need hardly be said that the most rigid aseptic precautions were used—*i. e.*, preventive

measures. The instruments were carefully cleansed with a nail-brush, soap, water, etc., the night before, wrapped in a clean cloth, and the package only opened to place them in recently distilled water. The utmost precaution about the assistants' hands, nails, clothing, and previous attendance upon contagious diseases, or the performance of post-mortem examinations, was observed. The silk used as ligatures had been thoroughly boiled in pure water—in fact, although antiseptics were not directly used, asepsis was secured. *February* 25, 1886.

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20. *Dermoid cyst of the ovary.*

Exhibited by Dr. T. R. Neilson.

This specimen I removed on April 26th last, from a woman, æt. 30 years, married, and the mother of two children. She suffered for some time before I saw her from frequent micturition, and symptoms due to pressure of the uterus to the left forward and downward. Upon examination I found the bladder, or, rather, the dilated urethra, pushed by superincumbent weight, nearly between the *labia*, and having a capacity of not more than an ounce and a half. The uterus bore down on it from above, being just behind the pubic symphysis and directed to the left side.

With the assistance of Dr. Howard A. Kelly, and at his private hospital, a thorough examination was again made, and the existence of an abdominal tumor, as a cause, and the above-named symptoms, as its effects, clearly established. I first performed Emmet's "button-hole" operation to take up some of the overstretched vesical mucous membrane in the hope of doing some good to the urethrocele. A week later I performed ovariotomy, with Dr. Kelly's assistance, and obtained this specimen.

I shall not here say anything about the operation itself, that being a matter of purely surgical importance, and of which I shall have something to say elsewhere. The patient made an uninterrupted recovery. The tumor, before it was opened, was tensely stretched, had no adhesions, was about as large as an adult's head, and sprang from the right ovary, broad ligament, and cornu of the uterus, by a broad base or pedicle. It contained nearly two quarts of a soapy-feeling fatty substance, and a large tuft of hair. Its walls are thick and elastic, smooth on the outside, and on the inside smooth, also on the whole of one aspect, and on the

upper half of the other, while the remainder of that side has growing from it numerous tufts of black hair, and in the same neighborhood are three large and one or two smaller pieces of bone growing in the wall. Two of the large pieces look something like the upper and lower epiphyses of an infant humerus; the other seems flat and curved, with a compact layer and a cancellated one, something like a cranial bone. The small pieces of bone are so irregular in size and shape as not to resemble anything. Toward the pedicle of the tumor, which it was necessary to separate from it, before its removal could be accomplished, are several smaller secondary cysts, containing fatty matter, and developed, evidently, in the wall of the larger cyst. The remainder of the ovary itself is cystic in places. *June* 10, 1886.

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21. *Endometritis.*

Exhibited by Dr. B. Reed.

Mrs. G. B. B., æt. 48 years. For ten years has been having menorrhagia, and occasionally passed from the uterus small lumps of flesh similar to the specimens sent herewith. Four years ago I first examined her *per vaginam*. Found uterus enlarged to three and a half inches in length; anteversion and marked unilateral laceration of the cervix. Did not dilate the cervix. Hemorrhage was severe every month—sometimes exceedingly exhausting. During the past two or three years the uterus has been scraped repeatedly with the dull curette. About two years ago Dr. Noeggerath, of New York, saw her with me, and noted some induration of the anterior wall of the uterus. During my absence in Europe, last winter, Dr. Philip Marvel attended her, and Dr. Julius Kæmmerer saw her with him. They did one thorough curetting, which was followed by severe inflammatory reaction, to which she nearly succumbed. Improvement followed each curetting, and there has sometimes been an absence of excessive hemorrhage for two months. The cavity of the uterus now measures from four to four and a half inches. On the 11th of the present month (August, 1866), two or three ounces of soft masses, like the accompanying specimen, passed from her after an extensive hemorrhage lasting for several days. I made styptic applications, and she has since got about the house again.

She has considerable pain in the sacrum at times, but not violent nor

very persistent. She is anæmic, but not cachectic in appearance. There has been at times a thin, watery, and somewhat offensive discharge from the uterus, but this has not been persistent. *September* 9, 1886.

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22. *Specimen from a case of carcinoma of uterus, with secondary deposits in omentum, peritoneum, liver, left pleura, and lung.*

Exhibited by Dr. T. R. Neilson.

This mass (specimen shown), which is about the size of a hen's egg, grew in the omentum of a patient of mine who died of cancer of the uterus, with secondary deposits, as will be described. Dr. William Osler saw the case with me in consultation, and also kindly aided me at the autopsy.

The history briefly is as follows: A maiden lady, æt. 46 years, tall, of slender build, but always enjoying good health, began to lose flesh and to suffer from occasional pain in the right iliac region about five months before I saw her, which was about the 1st of November last. Emaciation soon followed these early symptoms, the latter of which, the pain in the right iliac region, grew more frequent and more severe, and in September an eruption of eczema papulosum appeared in the left infraclavicular region, causing distressing itching. Her appetite and strength continued to fail, and she took to her bed about the middle of October. About two weeks later I saw the case for the first time, finding marked emaciation, the distressing eruption above-named, and also that the patient complained of a severe pain in the left chest, worst just below the breast, and extending across the back to the right axillary line, and, in addition, pain extending up the neck in the region of the nerve plexuses, and outward toward the shoulders. Both cough and fever were absent. The pulse was soft and frequent, 104 to 130.

Physical examination of the thorax revealed the presence of effusion in the lower part of the left pleural sac, with dulness on percussion, and impaired respiration over the whole lung. Palpation of the abdomen showed the uterus to be enlarged to the size of a very large pear, and in the right iliac region close to Poupart's ligament was the mass, which I show, and which was almost immovable. (Dr. Osler confirmed the results of this examination in consultation.) Some diarrhœa set in, and the patient died three weeks later, of exhaustion. The family stated that a grandfather had died of cancer of the stomach, but knew of no other case.

Autopsy.—On opening the abdomen, the omentum was found studded with small whitish masses, with here and there larger ones, the size of small beans, and in the right iliac region and attached by a band to the summit of the bladder, the one I show you, the size of which is about that of a hen's egg. The peritoneum was everywhere studded with miliary and slightly larger nodules. The uterus, which was about the size it attains in the fourth or fifth month of gestation, was thin and fluctuating, above the cervix (which was dense and thickened), and upon cutting it a quantity of thick tarry blood, having the odor of prussic acid, escaped; the cervix was plainly the starting-point of the disease, and its canal was obliterated, the tarry blood being retained menses. The liver was studded with many growths of the size of a pea, and one or two larger than a cherry. The left pleura was studded densely with miliary growths and some larger ones on both surfaces, and its cavity contained about f℥iv–vj of fluid. The lung was the seat of many deposits, in size from that of a pea to that of a pigeon's egg. Microscopical examination confirmed the diagnosis of scirrhus. *December* 9, 1886.

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23. *Microscopic specimens from a tumor of the vagina.*

Exhibited by Dr. G. E. de Schweinitz.

The tumor, the slides from which I exhibit, had the following history: A young woman, æt. 20 years, unmarried, had an abscess of the vagina, situated in the right labium. This opened on the mucous surface, and at this point there appeared a fungus-like mass, resembling a bunch of granulations. This was removed by the attending physicians, Drs. Ziegler and Wharton, and submitted to me for microscopical examination. The structure of the growth under the microscope is the following: 1. A layer of densely packed granulation cells, through which are scattered numerous giant cells; 2. A stratum resembling in structure a mucous gland, the oval and round alveoli of which are lined with columnar epithelium; but in places this epithelium has begun to proliferate, so that the appearances are those of a malignant growth; 3. Finger-like processes, composed of a basis of connective-tissue and capillary supply, capped with an abundance of epithelium, as in the ordinary papilloma. It seems to me that the natural history of this tumor is this: a papilloma formed in the neighborhood of gland tissue, perhaps the glands of Bartholin; abscess formation followed, and its

evacuation was succeeded by a bunch of granulations. The section has included the granulation tissue, the remains of the gland, and part of the original papilloma. *February* 10, 1887.

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24. *Uterus unicornis.*

Exhibited by Dr. F. A. Packard.

Specimen removed from the body of Ann McM., white, æt. 50 years. Patient died of cerebral softening at the Pennsylvania Hospital. She was, during the whole of her stay in the hospital, in such a stupid condition that but very contradictory and unsatisfactory answers were obtained concerning her past history, so that, very unfortunately, no history of her menstrual life could be obtained from her.

Among other interesting conditions found at the post-mortem examination, were the following anomalies of the uterus and its appendages:

The left Fallopian tube was extraordinarily long, its fimbriated extremity reaching up into the left iliac fossa, and the ovary being situated at a point two-fifths of the distance from the uterine cornu to the fimbriated extremity of the tube. The left ovary was small, firm, and of smooth surface, without the usual puckered appearance and with a well-defined mesovarium. The right Fallopian tube was also long, the fimbriated extremity being curled around and adherent to the right ovary, which organ presented the same characteristics as the left. The uterus is very small, and is composed of a cervix, a moderately large left cornu, and an exceedingly small rudimentary right cornu, with a wide notch between the two latter. The right cornu is simply represented by a nipple of uterine tissue about three-eighths of an inch in length, and of about the same diameter as its Fallopian tube, whereas the left cornu is of almost the size one would expect to find in a cleft uterus with two perfect horns. After careful examination, no trace of an os could be discovered. The woman was unmarried, and, presumably, had never borne children. There were, however, found unmistakable evidences of cicatrized gummata in her liver, and the kidneys both had small gummata present in them. She had also marked atheromatous changes, localized entirely in the circle of Willis and its branches. On opening the uterus, the cavity was found to be very small and filled with dried mucus, or, possibly, shed epithelium. No trace of a vaginal outlet could be found. It would, therefore, seem that this was a uterus unicornis without an os. *May* 12, 1887.

25. *Salpingitis followed by peritonitis.*

Exhibited by Dr. A. H. P. Leuf.

The specimen to be presented by me this evening was removed from the body of a patient of our President, Dr. J. C. Wilson. Following is an account of the autopsy.

The body was well nourished and almost fully developed. It appeared to be that of a person of about 17 or 18 years of age. The complexion was dark. Pubic hair was abundant and mammæ well developed. Abdomen was distended and tympanitic. The omentum was perfect, covering all the viscera below the transverse colon and enveloping them on either side. Its small vessels were well injected, as were also those of the abdominal wall. The injection was most marked below. The omentum was adherent to the fundus and back of the uterus, to the left Fallopian tube and the sigmoid flexure and upper end of the rectum. The mesentery was injected, as was also the small intestine. It contained many enlarged lymphatic glands; some being fully two centimetres in length. Greenish-yellow pus was found in moderate quantity in the bottom of the pelvis. Both ovaries, Fallopian tubes, and infundibulæ, with fimbriated extremities, were intensely congested and markedly œdematous; the left were worse than the right. They were deep purple in color and parts of the left tube looked as though they would soon have become gangrenous. The peritonitis extended up as high as the diaphragm, being perihepatic and perisplenic. Both ovaries were soft, slightly larger than normal and multicystic. The intestine contained yellowish muco-pus and showed marked enlargement of its lymphoid follicles. Throughout its whole extent the solitary glands were easily discernible. Beginning in the lower end of the duodenum, the agminated glands showed a decided increase in size. They were mostly very pale, but here and there showed marked injection. In the lower part of the ileum a few had begun to ulcerate and so also some of the solitary glands. At the ileo-cæcal valve there were several round ulcerations, one of an irregular outline due to a coalescence of several round ulcers. Judging from the severity of the tubal and ovarian inflammation as compared with the general peritonitis, and the latter being more severe as it neared the pelvis, together with the history of the case, makes it fair to conclude that the focus of the whole trouble was in the left ovary and tube.

One case of this kind, I recall distinctly, that came under my obser-

vation in 1882. The girl was 11 years of age, but not unduly developed or over-grown. She was ill for about ten days, beginning with a menstrual discharge, subsequently developing peritonitis, which increased in severity until it landed her with the great majority.

Another case, in which I officiated after death for Dr. Cruickshank, of Brooklyn, was that of a single woman, æt. 20 years. She was menstruating and had to take to bed. In a couple of days she developed peritonitis, which ended the matter in a couple more days. I found in this case also a marked peritonitis with intensely inflamed tubes and ovaries, the right ovary, in fact, having half sloughed away. It seemed to me at the time, and does still, that the usual ovarian congestion accompanying menstruation had become so intensified, from exposure or some other cause, as to lead to acute ovaritis and gangrene, and that the inflammation extended, especially after the detachment of the slough.

In looking hurriedly over my post-mortem notes, I find two cases of death in girls of 11 years, due to peritonitis, but not of genital origin. Also a case in a boy 8 years of age. *May* 12, 1887.

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c. MALE ORGANS OF GENERATION.

26. *Tumor of the testicle.*

Exhibited by Dr. G. E. DE SCHWEINITZ.

About two years ago, the patient from whom this specimen was removed, sought medical advice on account of a sore upon his penis. This proved to be a hard chancre, and in the course of time secondary symptoms appeared, and the patient passed through the stages of a mild syphilitic attack. Under the prolonged use of mercury and the iodide of potash, the patient apparently regained his wasted health and discontinued his visits. Five months ago he again consulted his medical adviser on account of a painless induration of the testicle. Because of his previous history the diagnosis of syphilitic sarcocele was given, constitutional treatment ordered, and the patient advised to give up his business temporarily and go abroad. To this he consented, went to the Continent to one of the spas, and put himself under the care of a physician. This gentleman concurred in the diagnosis made in this country, and continued by the use of anti-syphilitic remedies to try to reduce or remove the swelling of the testicle. As time progressed, it was noted

that not only had no improvement taken place, but the growth of the tumor was increasing. The physician in charge, now strongly suspicious of the true nature of the malady, advised the patient to return home and again consult his American physician. This he did, and it was found that the tumor had markedly increased in bulk, had become more painful, and besides there was some inguinal involvement. The patient was depressed in spirits, his nutrition below par, his features cachectic. Speedy extirpation of the growth was advised and accomplished. The resulting specimen, which is before you, was sent to me for microscopical examination.

Sections of the growth reveal a stroma of young often spindle cells, interspersed with variously shaped collections of cells, closely resembling typical cancer cell-nests. The cross-sections of the seminiferous tubules exhibit themselves often in their normal appearance, but quite as often either in the beginning stage of active cell proliferation or else when the cells have actually passed the boundary and have passed out into the surrounding tissue to form or help to form the areas of cell aggregations before described. The growth is either a medullary sarcoma or, more properly, an admixture of, the sarcoma and carcinoma type. Even at the time of operation the involvement of the spermatic cord was noted, and a short time later palpation revealed secondary deposits in the abdominal cavity. To these complications the patient succumbed about one month after the operation.

No post-mortem examination was allowed. The points of interest in this exhibition are easily seen: the previous clinical history, the natural early mistaken diagnosis resulting from it, and the rapid return of the growth. *December* 10, 1885.

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27. *Epithelioma of scrotum.*

Exhibited by Dr. H. R. Wharton.

This growth was removed by Prof. Ashhurst, from the scrotum of a man, æt. 45 years, who was employed in the retort-house at the gas works. The growth is probably an epithelioma, and is of interest in connection with the history of the patient's employment, as it represents that form of epithelial cancer which is so commonly seen in England, to which the name of soot or sweep's cancer is given; although it differs in no respect from the ordinary form of epithelioma. *March* 24, 1887.

28. *Two cases of malignant disease of the testicle.*

Exhibited by Dr. G. McClellan.

Case I.—The patient's age was 65 years. The growth was removed by Dr. W. L. Atlee. It was a sarcoma containing a large cyst, involving the epididymis and cord. Death occurred within a week after operation.

Case II.—Patient's age was 27 years. The growth appeared to have followed a blow upon the testicle. It was a sarcoma involving the entire organ. Recurrence has not taken place. *May* 26, 1887.

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d. MAMMARY GLAND.

29. *Carcinoma of the breast; lipoma.*

Exhibited by Dr. J. H. Musser.

Eight years ago Mrs. B. received an injury in her left breast, succeeding which she almost constantly experienced a pricking or tickling sensation in the organ. Five years ago she distinctly noted a lump the size of a duck egg, hard and painless. She consulted me in 1883. The breast was large, hard, lobulated, and large vessels were seen coursing over its surface. The nipple was retracted. One lobule was congested on its surface, of a bluish hue. This ultimately became the seat of ulceration. The ulceration from this point extended over the outer surface of the half of the breast and was frequently the source of bleeding while a semi-purulent discharge always exuded from the surface. Fetor accompanied the ulceration, but pain was never experienced. The edges of the ulcer were thickened.

When first consulted a few enlarged glands could be felt in the axilla. Subsequently a large mass developed there and the chain of glands from the breast became involved. No œdema of the arm resulted. No other lymphatics were involved and no secondary internal deposits could be detected.

A large lipoma the size of a fœtal head, which was present since 1840, was also observed on the back of the arm of this patient. Accompanying the emaciation this tumor lessened to the size of an egg.

The patient was 75 years old, of excellent habits, and great activity. She had considerable care. Her food was not of any special character. During the last year of her life quite frequently Mrs. B. had attacks of excruciating pains in her leg bones and the left parietal bone. The tibiæ would be tender; the portion of the skull which was the seat of pain seemed to increase slightly in prominence with each attack and was tender on percussion. The attacks were accompanied by fever. Large doses of anodynes mitigated the pain, which was worse at night; almost immediate relief, however, would be given by the use of iodide of potassium. There was an absence of syphilis as far as could be determined in herself or of inherited syphilis in her children. There was no evidence of brain tumor or of gummata. The patient died of marasmus.

No autopsy could be obtained, but permission was given to remove the breast, which is on the table. The breast is about five times the size of its fellow, weighing three pounds at least. The large, flat, ulcerated surface is quite retracted. The nipple is atrophied and retracted. Portions of the gland structure seem to be free from malignant involvement.

Microscopical examination of the sections made from one of the indurated lobules showed the histological characteristics of scirrhus.

November 12, 1885.

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30. *Supernumerary mammary glands and nipples; three cases.*

Exhibited by Dr. W. A. Edwards.

That only about one hundred cases (105, Liechtenstern) of polymastia have been recorded will at once appear as the *raison d'être* of this brief communication. A somewhat unusual opportunity for the examination of patients in hospital practice and in the examination of recruits, has convinced the writer that these additional mammæ are not, at least in this country, of very frequent occurrence. It is proper, however, to add that Dr. J. Mitchell Bruce is of the opinion that the number of cases seen by practitioners and not reported must be very great. It becomes somewhat interesting to look into and, if possible, arrive at the cause of these abnormalities. Laycock, in endeavoring to explain the existence of the accessory glands, thinks that the mammæ are simply enlarged and highly developed sebaceous glands, and may make their appearance in any part of the body.

I might cite as a possible example of this theory, Klob's case of an

accessory mamma growing on the left shoulder of a man, on the prominence of the deltoid.[1] It was a true mammary gland, and had a nipple about a quarter of an inch in length, the entire gland being about the size of a walnut.

In turning to the morphological significance of these cases, we must consider the character of the mammary glands in the different orders of mammalia, remembering that in the marsupialia and the monotremata the glands possess no nipple, their orifices being mere scattered pores in the skin.[2]

In the Simiadæ, from the anthropoid apes to the marmosets or arctopithecini, "the teats are only two in number," and they are pectoral.[3] In the lower section of the Primates, the Lemuridæ, "sometimes there are one or two pairs of teats on the abdomen in addition to the ordinary pectoral pair."[4]

In the aye-aye (Cheiromys Madagascariensis) "there is but one pair of nipples, situated about one and a half inch in advance of the vulva, and one inch apart."[5]

We are all familiar with the vulvo-pectoral teats of the common carnivora and the inguinal udder of the ruminants. In the Pteropodidæ (fruit bats or flying foxes) and the flying "lemur," Galeopithecus, we find auxiliary teats.

So if we desire to explain the presence of supernumerary mammary glands in women by the reversion theory, we see that there are numerous places where the accessory organs may grow; yet, as we will see later, the recorded cases seem to differ in their site from all morphological laws.

That our species present the pectoral position of the teats does not show any special superiority, as the glands are found in the same position in the bat, dugong, manatee, elephant, sloth, and the ant-eater.

We do not find the number of teats always in relation to the offspring; as, for example, the rat has twelve, whereas the guinea-pig has but two; again, the tenrec (centetes), an animal somewhat like a hedgehog, has twenty-two nipples, and Owen tells us that in the European hedgehog there are ten, ranging from the pectoral to the inguinal region, adding that the thin, flat mammary gland of this insectivorous animal seems to form a continuous stratum.

Before leaving this part of our subject, it may be interesting to note

[1] Zeitschrift der k. k. Gesellsch. der Aerzte zu Wien, 1858, No. 52, p 815.
[2] Prof. W. H Flower, LL.D., F.R.S., and G. E. Dobson, M.A., M.B.
[3] Huxley's Manual of the Anatomy of the Vertebrated Animals. [4] Huxley, Ibid.
[5] Prof. Owen, The Anatomy of the Vertebrates, vol. iii.

that Professor Owen[1] describes a female orang-outang with an accessory nipple on the left side, below the normal one and of smaller size.

The case that I am about to record was noted in the spring of 1884, in company with my student, Dr. Thomas D. King, of Springfield, Ohio.

Case I.—John J. B., æt. 36 years, a healthy, well-developed man, came under my care for subacute alcoholism, and while examining his chest for impending pneumonia, an accessory nipple was noted on the right side almost on a line with and three inches below the normal one, similar in size, presenting an areola with glandular nodules and a few stiff hairs. There was no discharge from either the normal glands or the accessory nipple; it had never caused the patient any inconvenience, beyond a certain hesitancy and delicacy in confessing the abnormality.

In conversation with my patient, I learned that his sister presented the same abnormality, and an interview was arranged.

Case II.—Mrs. N. M., æt. 38 years, mother of four children, the youngest a nursling. The woman was a perfect specimen of the healthy Irish-American female that we so frequently meet.

The accessory breast in this case was on the left side, normal in appearance, somewhat smaller than the other two, but was functionally active with a good flow of milk from a perfectly normal nipple; during my visit the child would nurse indiscriminately from any of the breasts. The woman informed me that she had nursed all of her children in this way. I learned that the supernumerary gland developed coincidently with the others, becoming active as they did and again physiologically barren as their secretion retroceded.

These two cases, and the two reported by Handyside,[2] open an interesting field in the study of the inheritance of the abnormality. In no case recorded by Bruce (Ibid.) was heredity at all clear. Liechtenstern could find no illustration of heredity among his own cases, but remarks that inheritance was indicated in 7 of the 92 cases recorded in literature.

All the instances of supernumerary nipple that came under observation were situated on the front of the trunk, below the level of the ordinary mamma and somewhat nearer the middle line, but unfortunately for those adherents of the theory of morphological explanation of these cases, abdominal nipples are extremely rare. Bruce, however, describes two cases where one of a pair of nipples was distinctly below the costal cartilages.

[1] Anatomy of the Vertebrates, vol. iii. p. 730.

[2] Notice of Quadruple Mammæ, the lower two rudimentary, in two adult brothers, by P. D. Handyside, M.D., etc., Journ. of Anat. and Phys., vol. vii. p. 56, 1873.

Liechtenstern's conclusion, from his own experience and from the recorded cases, is that "accessory nipples and breasts occur in 91 per cent. of the cases at the front of the thorax; and in 94 per cent. of these below the normal." In one of Liechtenstern's cases, the nipple occurred in the axilla, which is an extremely rare situation. All observers agree in stating that additional mammæ or nipples are most frequently seen on the left side. Liechtenstern endeavors to account for this by the well-known fact that the left mamma is, as a rule, more developed than the right, and that mothers use the left more than the right in suckling. He is of the opinion that the same relation exists in the child and virgin.

As to the frequency of this abnormality, I may mention that in Bruce's investigations of 3956 persons, 61 cases of supernumerary mammæ or nipples were found, being 1.54 per cent. Of these cases 51 were simple cases, presenting a single supernumerary nipple or mamma, 10 were multiple, presenting more than one supernumerary nipple.

A perusal of Mitchell Bruce's exhaustive article would lead one to conclude that supernumerary nipples or mammæ must be much more frequent in England than they are in this country, as out of 315 persons taken as they come and go in a dispensary service, he found that 287 individuals presented no supernumerary nipples or mammary glands; 24 individuals presented one or more supernumerary nipples or mammæ; and 4 individuals presented appearances which were regarded as doubtful instances of the abnormality. A representation of this result in percentages would be as follows: 91.1 per cent. presented no supernumerary nipple or mamma; 7.619 per cent. presented one or more supernumerary mammæ or nipples; and 1.27 per cent. possessed what was doubtfully regarded as the abnormality. This is certainly a high rate of frequency, even above that of Liechtenstern, who places it at "1 in 500 persons at least." As to the relative frequency of the abnormality in the sexes, it appears from the above statistics, which are undoubtedly the best that are available, that supernumerary nipples occurred very nearly twice as frequently among men as among women; this conclusion also agrees with Prof. Liechtenstern's result. It will be readily understood that definite statistics are extremely hard to formulate in these matters, as some cases will undoubtedly be overlooked unless attention is particularly called to the mammary regions. Again, some of these supernumerary nipples may escape detection on account of their want of resemblance to the normal standard; for example, the additional nipple may be wanting in the areola, the follicles, the marginal hairs, or, again, the papilla may be poorly developed, as may also be the terminal

depression or opening. We may be misled by a hair springing from the summit of the papilla, as in one of Bruce's cases.

Before leaving the subject, it is, perhaps, well to mention some cases of supernumerary mammæ in unusual situations.

Dr. Fitzgibbon[1] records the case of an individual with double and symmetrical supernumerary nipples below and within the normal, together with two pigmentary deposits, one on either breast higher up.

Accessory mammæ, with nipples, have been found in the axilla,[2] and to Liechtenstern also belongs the credit of describing two cases of dorsal mammæ; his references, quoted by Bruce, are as follows: 1. Ch. F. Paulinus (*Observat. Medico-physic Select.*, in the *Miscell. Curios. Academ. Med. Phys. Nat. Curios.*, dec. II. ann. iv. page 203, Appendix); 2. Joh. Otto Helbig (*De rebus variis indicis*, Obs. 194, quoted in *Miscell. Curios.*, etc., dec, II. ann. ix. and x. p. 456, ii.).

Again must we credit Liechtenstern with showing the fallacy in the statement of the existence of inguinal mammæ. He has pointed out the fact that the idea has arisen by careless reference to Roberts's case (see Luschka, *Anatomie des Menschen*, Band I. Abtheil. 2) of a woman who had an accessory mamma on the outer aspect of the left thigh, four inches below the trochanter; it was accidentally discovered by the patient's child, who one day attempted to suck the nipple.

Dr. H. L. Turney[3] reports a case of additional mammary gland in the right axilla; it was nippleless, but secreted milk freely from several orifices after each confinement.

Dr. Cohn presented a patient before a meeting of the Berliner Medizinische Gesellschaft, in February, 1885, with an accessory mammary gland in the left axilla, from which milk dribbled away. There was no nipple. Mr. A. H. E. Cameron[4] also records a case of left axillary mammary gland without nipple; and Mr. Bickersteth informs him that he has seen two similar cases of nippleless tumors bearing the characters of mammary glands.

The following case occurred in the practice of Dr. H. R. Wharton, of Philadelphia, to whom I am indebted for the notes:

Case III.—Mrs. S., æt. 27 years, in her third confinement, called my attention to a supernumerary nipple on the right breast, situated about three inches below the normal nipple, just at the edge of the mammary gland. It was smaller than the normal nipple, had a well-developed

[1] Dublin Journal of Medical Sciences, vol. xxix. p. 109. February, 1860.
[2] Liechtenstern, with illustration.
[3] Nashville Journal of Medicine and Surgery, April, 1883.
[4] Journal of Anatomy and Physiology, 1879, vol. xiii. p 149.

areola, contained milk, and the child could use either indiscriminately. Before her first confinement she thought it was a mole, but at this time it became enlarged and contained milk which escaped freely from it at the time I saw her.

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The Medical News (editorial), November 28, 1885.

January 14, 1886.

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31. *Large adenoma of breast.*

Exhibited by Dr. J. M. Barton.

Ellen W., æt. 17 years, colored, was admitted to Jefferson College Hospital April, 1887. Her left breast began to increase one year ago, until at the time of removal, April 13th, it measured twenty-seven inches in circumference. It was distinctly pedunculated, its circumference at the base being only twenty-one inches; its outline was regular, its surface perfectly smooth. The superficial veins were greatly enlarged, some being as large as the little finger; the nipple was retracted and surrounded by

an eczematous eruption, probably due to pressure, as this was the most dependent part of the growth.

On palpation I found no distinct tumor in any part; it was more dense than the normal breast, and in all parts it felt as though an irregularly coiled small, hard rope were between the fingers. There was no sensation of fluctuation, little if any increase of heat; it was not adherent to the skin nor to the deeper structures; there was no glandular enlargement in the axilla, the right breast was a fully developed normal breast.

This is quite a rare form of growth, and is described by most writers as "hypertrophy." It occurs most frequently in females about the seventeenth to twentieth year; both breasts are generally enlarged. In 34 reported cases both breasts were enlarged in 22 cases, the right only in 7, the left only in 5 cases.

On section it presents a grayish, homogeneous surface entirely free from fat. On microscopical section it is a fibro-adenoma, presenting, however, much less stroma than is found in the small adeno-fibroma so common in the female breast about the age of forty to forty-five years, and very much less than in the normal breast, the main increase being in the glandular elements.

I removed the breast April 13th of that year; the wound was dressed antiseptically; it healed without suppuration or any rise in temperature. The patient was discharged three weeks after the removal, with the wound entirely healed. *June* 26, 1887.

VI. THE NERVOUS SYSTEM.

1. *An imbecile brain.*

Exhibited by Dr. F. X. Dercum.

The patient, from whom this specimen was obtained, was a male, and an inmate of the State Hospital at Norristown. He was aged about 30 years, and was an epileptic imbecile.

On removing the calvarium, which exhibited nothing abnormal, the dura was found somewhat thickened, but not adherent. On puncturing the latter, about four or five ounces of cerebro-spinal fluid escaped. The pia mater was adherent in a few areas; the ependyma was reddened and injected, and the velum interpositum was thickened.

The convolutions were remarkable in some respects, and presented the following peculiarities:

They were, for the most part, unusually small, while the sulci were exceedingly shallow. They were best developed in the parietal region, but even here they were decidedly abnormal. Anteriorly the frontal convolutions were outlined, but the fissures constituted little more than shallow grooves. This was especially the case in the right hemisphere, in which also the third frontal convolution was very poorly developed, the operculum absent and the temporal lobe exceedingly small. The island of Reil was thus freely exposed. In the left hemisphere the island was, on the contrary, almost completely covered.

The fissure of Rolando pursued an average course, with the exception that in both hemispheres it ran directly into the Sylvian. The interparietal fissure presented nothing unusual in its course through the parietal region, but when it reached the occipital lobe it became shallower and was lost upon the general surface. It extended back a little further and was a little deeper upon the right than upon the left hemisphere. The two temporal fissures were quite shallow. The first convolution on the left side was exceedingly narrow; the second and third were somewhat better developed. The supra-marginal lobules of both sides were quite small; this was also the case with the angular gyri,

especially the left, which was *excessively* small. It was exceedingly narrow and *angular* in shape.

It was in the occipital lobes, however, that the most interesting points were noted. These lobes were remarkable for the smoothness of their surfaces. On the lateral aspects were noted only the short and shallow impressions of the interparietal fissure, and a shallow transverse groove corresponding possibly with the normal longitudinal occipital fissure.

Upon the mesial surfaces, the shallowness of the fissures was in keeping with those already described. The calcarine fissure was exceedingly short and in both hemispheres, extended directly into the hippocampal fissure. The gyrus hippocampi was present on either side, but so small as not to rise to the surface. The posterior end of the calcarine was bifurcated in the right brain and simple in the left. On both sides the cuneus was much distorted by the aberrant course of the fissure parieto-occipitalis. In the right hemisphere the fissure *curved forward* and ran for a short distance parallel with the fissure of the corpus callosum, then abruptly turned upward and terminated upon the lateral surface. A comparatively well-marked superior *pli de passage* was present. Instead of making an acute angle of 50 or 60 degrees with the calcarine fissure, it made an obtuse angle of about 120 degrees. In the left hemisphere the parieto-occipital made a right angle with the calcarine and ran perpendicularly upward to end in a bifurcation upon the lateral surface. On this side, also, a large superior *pli de passage* was found.

The brain can only suggest atavism in a remote sense. It was essentially a case of arrested development, the arrest being most marked in the frontal and occipital regions. The occipital lobes are anomalous in the extreme, the distortion of the cuneus and parieto-occipital fissures being the main features. Furthermore, the excessive quantity of cerebro-spinal fluid and the condition of the pia and lateral ventricles tell us that we have a brain to deal with which is essentially diseased.

Microscopical examination.—A small fragment was removed from the paracentral lobule of the left hemisphere and sections cut. It was extremely difficult to separate the cells into layers. In the upper portion of the cortex the cells, though numerous, were rounded or irregular in shape; in the lower portion the cells were comparatively few in number, and pyramidal cells were the exception; only one or two large cells like those usually found in the normal cortex in this position were seen. The pia appeared to be thickened, but the walls of the vessels showed no decided change. The amount of neuroglia was relatively large.

The examination of the other organs of the body revealed old pericardial adhesions and chronic inflammation and ulceration of intestine. Nothing else worthy of note was observed. *December* 10, 1885.

2. *Two cases of abnormality of the brain.*

Exhibited by Dr. H. F. Formad.

Case I.—Boy, æt. 9 years. He had always been healthy, and of fully average ability, death resulting from an accident. There was an entire absence of falx cerebri, the two hemispheres being connected by a bridge of brain substance above the corpus callosum. There was nothing else abnormal.

Case II.—As a young child the intellect had been normal. After a sunstroke imbecility came on. The brain was markedly asymmetrical, the left cerebrum being fully twice as large as the right, with an entire difference between the two in the topography of the convolutions. On the contrary, the right lobe of the cerebellum was twice the size of the left. The skull was shaped to suit the brain. *December* 10, 1885.

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3. *Case of meningocele.*

Exhibited by Dr. C. M. Wilson.

The subject from whom the tumor was removed was an outpatient of the Philadelphia Lying-in Charity. It was the youngest of four living and healthy children, born fourteen days before our attention was especially called to the case. Very curiously, the child born two years before this one had a nævus in the left temporal region. At the time of its birth the gentleman of the class to whom the case had been assigned noticed a tumor, the size of an English walnut, over the posterior fontanelle. It was a fluctuating mass, and at one time it appeared as though suppuration was taking place.

Forty-eight hours before death the child was seized with convulsions, rapidly succeeding each other. They continued until the end. Trismus was well marked.

The tumor had shrunk considerably in size. In its removal post-mortem, it was included in an elliptical incision. The large membranous space, constituting the posterior fontanelle, was also excised. The sagittal and lambdoidal sutures were imperfect, being separated by membranous spaces, the former three-quarters of an inch, and the latter half an inch in width. The tumor was found to be narrowed at its

base, having a small pedicle, which passes through an opening in the fontanelle. The outer layer of this is continuous with the dura mater. Within this there is a cord, having the appearance of organized lymph. A probe passed from the dura into the cyst. It was filled with a caseous matter.

There were about two ounces of a sero-purulent effusion.

December 10, 1885.

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4. *Intracranial hemorrhage; record and classification of 143 consecutive fatal cases observed in medico-legal practice and in private autopsies in the city of Philadelphia.*

Exhibited by Dr. H. F. Formad.

Dr. Formad made some remarks on the different forms of intracranial hemorrhage he had observed, and presented a number of specimens in illustration. There exist, he said, no well-defined descriptions of the various forms of intracranial hemorrhage with reference to their etiology, and no classification that can be of any service in medico-legal examinations, and he suggested the classification and observations presented below, as particularly adaptable for the latter purpose. These records may also be of use to the surgeon who wishes to trephine. The observations were sufficiently numerous to warrant conclusions of importance for various ends. Dr. Formad regretted that he met his cases on the autopsy table only, and hence was unable to offer clinical details. In some cases the history was far from being reliable, and in a few the history was left to conjecture from the surroundings, the circumstances of the case, and the appearances and general lesions of the body.

Dr. Formad wished to give credit to his colleague in the Coroner's work, Dr. R. R. Stewart, whose assistance in these observations he enjoyed, and also to his assistants Drs. A. J. Plumer and George H. Chambers. Dr. Formad's cases of intracranial hemorrhage embrace the results of various kinds of head injuries (traumatic), and cerebral apoplexies (idiopathic hemorrhages) as well. By the term cerebral apoplexy, he meant exclusively hemorrhages in the brain, and did not refer to "congestive," "serous," "embolic," or "nervous" apoplexies and other affections which tyrannic nomenclature has thrown together under the term of "apoplexy." Idiopathic cerebral apoplexy should also be strictly separated from traumatic cerebral hemorrhage; this is not done by many writers.

The latter can be further subdivided into very well defined sub-varieties of hemorrhages, each etiologically distinct, as will be shown below. These observations will clearly show the necessity and usefulness of a classification such as suggested. There should never be any reasonable doubt at the post-mortem table as to whether a cerebral hemorrhage was due to disease or to injury, or to both combined; nor as to whether a head injury preceded or followed an intracranial hemorrhage. Dr. Formad believed that the diagnosis between the conditions just stated is much more difficult during life. He had seen errors made by foremost surgeons and physicians, and in the best hospitals in this city. He had seen cases of apoplexy diagnosed as fracture, and trephined, and he had seen cases of fracture of skull unrecognized and untouched, left to die in the medical wards.

Upon the post-mortem table, however, there should be no difficulty in diagnosing such lesions in the present status of morbid anatomy as a distinct and well-defined science. After sufficient accurate observations have been made, and after seeing conditions repeat themselves, case after case alike, and having in the majority of instances cause and effect directly before the eyes—one should be enabled in disputed cases to make a post-mortem diagnosis which leaves but little room for doubt.

Dr. Formad divided the cases of intracranial hemorrhage observed by himself into the following eight different classes, the accounts being given here in abstract form.

Class I. 12 Cases.—Post-mortem appearance: One or more small hemorrhages in the fasciculi teres of the fourth ventricle; also ecchymoses in the lining of the other ventricles (no massive hemorrhage anywhere); cerebral substance as well as membranes intensely congested. Cranium much congested; drops of blood oozing from the cut surface and from the periosteum. Marks of contusion in scalp, with more or less ecchymoses noted in all the cases.

Histories: In two cases death was caused by electricity (details will be recorded elsewhere). Ten cases revealed histories of concussion of the brain from injuries to the head, without fracture of the skull. Death was rapid or sudden from shock in all these cases. Six were males and six females; the majority were young persons.

Class II. 23 Cases.—Post-mortem appearance: Massive hemorrhage (blood-clot) of various sizes found between pia and dura mater, location in the majority of cases about opposite to place of application of violence. In none of these cases was this meningeal blood-clot located immediately beneath the point of application of violence. Ventricular ecchymoses also usually found. Brain anæmic wherever hemorrhage

was large. Scalp injury well marked in some, in others less so, while in a few cases it was hardly noticeable.

Histories: Concussion from injuries to head without fracture in twenty-one cases; with fracture of skull in two cases. Death rapid (all within one hour) or sudden, either from shock or from compression of brain, the result of pressure from clot or from both. Fourteen males, nine females; ages varied.

Class III. 45 Cases.—Post-mortem appearance: Massive hemorrhage (blood-clots), usually quite large, located between dura mater and inner surface of fractured skull, and at the site of application of violence only. Brain anæmic.

Histories in all the cases revealed injuries to the skull from accidents, blows, or other violence, resulting in fracture of the skull. Death on the average ensued slowly from compression or anæmia of the brain, after the lapse of from several hours to several days (in a few cases from five to eight days). Thirty-eight males and seven females; majority young and middle-aged persons.

Class IV. 12 Cases—Post-mortem appearance: Massive hemorrhage, the same as in the preceding class, but occurring both at the site of application of violence, and also on the same point on the opposite side of skull; in the first, clot large between the dura mater and the skull; in the latter, usually a small clot in the arachnoid (between the dura and pia mater). At the site of the injury there was found a fracture of the skull in all these cases, so that they presented the combined features of Classes II. and III.

Histories in these twelve cases showed injury to skull, resulting in both fracture and concussion. Death was nearly instantaneous in all the cases. Nine were males and three were females; mostly young and middle-aged persons.

Class V. 9 Cases.—Post-mortem appearance: Punctiform and multiple hemorrhages into the tissue of the dura mater, which showed much thickening and many adhesions to inner surface of the skull from chronic pachymeningitis; great œdema of the pia mater. Brain congested but no blood-clots in either ventricles or substance.

Histories in these cases revealed former attacks of sunstroke, or old injury of the head. Death usually sudden, and usually following a fall or severe intoxication and excitement. Ages of deceased, between twenty and fifty; all males.

Class VI. 34 Cases.—Massive hemorrhage of various sizes into the brain substance proper, and thence into one or both of the lateral ventricles. Hemorrhagic softening of the brain substance of the corpora striata,

or their vicinity. No meningeal hemorrhage in any of these cases, though in some instances blood was observed to have leaked out from the inner ventricles through the transverse fissure and fourth ventricle, and some, usually liquid (post-mortem), blood was seen in the fissures upon the periphery of the brain, in the meshes of the pia and arachnoid. Brain substance in nearly all cases quite anæmic. Atheroma of cerebral vessels in all but three cases. Multiple small aneurisms of cerebral vessels noted in several cases. Chronic pachymeningitis in various degrees, in all but five cases. In some cases there were scalp injuries from secondary falls—*i. e*, falls that were the result of preceding cerebral hemorrhage. Many of these cases came under the Coroner's jurisdiction, on account of suspicion of violence.

Histories wherever obtainable, revealed either alcoholism, rheumatism, or syphilis; middle or advanced age. Twenty-six males and eight females. Death in the majority of cases ensued slowly. These were true cases of idiopathic cerebral apoplexy.

Class VII. 6 Cases.—Post-mortem appearance: Massive hemorrhage of various sizes below pia mater, into brain substance, without breaking into lateral ventricles. In one case the blood-clot involved the substance of the pons, and protruded into the fourth ventricle; in two cases it occupied the substance of the right parietal lobe, which was the seat of an abscess; in three cases the hemorrhage was multiple, in small foci, with localized hemorrhagic softening of the periphery of the brain substance in various places. In three cases there was atheroma, and in the other three fatty erosions of the cerebral vessels. Chronic pachymeningitis in all the cases. Brain substance congested.

Histories were those of former head injuries, alcoholism, and, probably, syphilis. Death rapid, following intoxication or exertion. Age advanced in all the cases; five men and one woman. Foul play had been suspected in all of these cases.

Class VIII. 2 Cases.—Post-mortem appearance: Massive meningeal hemorrhage below pia mater, combined with hemorrhage into ventricles; no visible hemorrhage into the brain substance. Atheroma and multiple small aneurisms of vessels of pia and of choroid plexus. Ecchymoses in scalp.

Histories of accidental falls. No previous symptoms of cerebral disease had been noted. Intemperate habits, injury of the head, leading to rupture of the diseased vessels, evidently had preceded the cerebral hemorrhage. The persons were at work (washing) at the time, when, nearly simultaneously with the concussion from the fall, death ensued. Both were women; ages 57 and 63 years.

Remarks and Comments.—A number of cases of intracranial hemorrhage, observed by Dr. Formad, he had excluded from the series above detailed. Several cases of compound comminuted fracture in which the skull, membranes, and brain, were partly pulpified into a mass, and hence the hemorrhage not satisfactorily made out, were excluded from the above records. He also excluded chronic cases of cerebral hemorrhage and those resulting in abscesses, or in a fatal lepto-meningitis or cerebritis after the patients had partly recovered from the immediate effects of the hemorrhage. Dr. Formad did not incorporate into these records another large class of intracranial hemorrhage, viz., that of infantile meningeal hemorrhage. Of these he had seen many in coroner's work. Meningeal hemorrhage is common in infanticides and fœticides, and even in stillborn children. Dr. Formad could not plausibly explain the frequency of meningeal hemorrhage observed in the stillborn, otherwise than by presuming that they were due to traumatic injuries from violence, or to compression of the cranium during protracted labor or when forceps had been injudiciously applied. He did not meet with cerebral hemorrhage in children, other than meningeal.

Returning to his classification, Dr. Formad stated that the quantity of the hemorrhage or the size of the clot in any case of intracranial hemorrhage appeared, in all the cases he observed, to depend upon the duration or the time that elapsed from the moment of injury to death. In cases where death ensued rapidly or instantaneously from shock, as in many fatal cases of concussion, or even in fractures of the skull followed by immediate death, as in falls from a great height, the bulk of the ante-mortem hemorrhage (the blood-clot) was remarkably small; or clots were occasionally entirely absent, and only a post-mortem oozing of liquid blood could be observed. When death is protracted for several hours, then the blood-clot is usually large, weighing up to six or eight ounces, and causing death by compression of brain. In some cases of fracture of skull the hemorrhage is probably delayed for several hours, or ensues but very gradually. Some patients are said to have walked around for many hours after the injury, and subsequently when the blood oozed out in sufficient quantity to compress the brain, they fell, became unconscious, and died in coma. In one case of this nature he had found a meningeal clot weighing eight ounces.

Dr. Formad observed that in the cases in which the intracranial hemorrhage was voluminous and death instantaneous, there was very little scalp ecchymosis; whereas scalp hemorrhage was more pronounced if the intracranial hemorrhage was small, and death delayed.

Further, Dr. Formad noticed that in no case where there was internal

cerebral hemorrhage, was there any meningeal hemorrhage when cerebral vessels were normal; and in no case of traumatic meningeal hemorrhage did he see any coincident ventricular hemorrhage (except small ecchymoses), unless there existed some old lesion of the vessels or the substance of the brain. It appears, however, that blood from the lateral ventricles may leak through the foramen of Monro into the third ventricle, and thence by the aqueduct of Sylvius find its way into the fourth ventricle; but Dr. Formad did not see in any one of the cases he observed that primary hemorrhage into the fourth ventricle ever extended to the lateral ventricles, probably on account of its rapidly fatal character.

A hemorrhage on the outside of an uninjured pia mater cannot reach the interior of the brain or the lateral ventricles.

The source of the hemorrhage is often very difficult to determine. In fracture of the skull the hemorrhage is often diploic; in concussion of brain the hemorrhage has for its source the vessels of the pia mater; in diseased conditions of the brain, or in atheroma of vessels, the small feeding branches of the middle cerebral and of the basilar artery bleed most frequently, and the blood breaking into the lateral ventricles forms clots in either one or both of the ventricles.

The view expressed by Dr. J. A. Lidell, in his treatise on apoplexy (N. Y., 1873, p. 113), that "meningeal hemorrhage not unfrequently occurs spontaneously as well as in consequence of the operation of violent causes;" and that (see page 120) "meningeal hemorrhage of spontaneous origin is not a rare affection," is unquestionably erroneous. Yet such "views" are quoted by writers on forensic medicine, and presented to juries in murder trials as facts.

Ill-informed or ignorant post-mortem examiners can set murderers free, or, on the other hand, create unnecessary court trials.

Conclusions.—1. Hemorrhages exclusively above the pia mater and above the dura mater—*i. e.*, on the outside of the brain—are always due to *traumatism* or *sunstroke*, provided a cerebral source for hemorrhage is excluded, and the cerebral vessels and membranes are not diseased.

2. Hemorrhage in the floor of the fourth ventricle is always traumatic, provided there are no blood-clots in the lateral ventricles, or any other part of the cerebral substance.

3. Hemorrhage exclusively below the pia mater, or in any part of the brain-substance, or in the ventricles (except the fourth), is always idiopathic—*i. e.*, is due to disease.

4. There must be a diseased condition of the cerebral vessels or substance in order to ascribe a hemorrhage to disease. There must be

traumatism (a fall or some form of violence) in order to induce hemorrhage in a normal brain.

5. The blood-clot in concussion of the brain is not found at the point of application of violence, but always somewhere about the opposite side of the brain, and always within the arachnoid—*i. e.*, between the pia and dura mater.

6. The blood-clot in fracture of skull is always found at the point of application of violence, immediately below, and always between the dura mater and the fractured part of the skull itself.

7. A blood-clot formed within the cranial vault is more favorable to the patient if due to fractured skull than if due to a mere concussion.

8. Only clotted blood and infiltration of blood-corpuscles into tissues indicate an ante-mortem hemorrhage; liquid blood is due to post-mortem oozing, and only stains but does not infiltrate tissues.

9. Severe bruises and cuts of the scalp may be seen in cases of idiopathic apoplexy where a sudden cerebral hemorrhage causes a person to fall.

10. In some cases it is impossible to decide by medical examination alone as to whether a head injury, and the resulting hemorrhage, is due to a fall or to violence.

11. External marks of violence may be invisible to the unaided eye in some cases of injury of the head or other parts, but are easily detected by the microscope, and readily distinguished from post-mortem spots. (See 8, above.)

12. The bulk of the intracranial hemorrhage stands usually in inverse proportion to the external scalp hemorrhage. *March* 25, 1886.

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5. *An unusual case of cortical hemorrhage with rhythmical contractions of left side of the body.*

Exhibited by Dr. T. D. Dunn.

Mary W., æt. 73 years, widow, came under my care January 23, 1886. She was suffering from what was considered congestive apoplexy. In a few days she had recovered sufficiently from the attack to be able to leave her room. She had always enjoyed good health, but there had been some mental impairment for several months. There was no perceptible cardiac disease; the arteries, however, were hard and rigid.

Before day on the morning of the 6th of February, the family was aroused by a peculiar shuffling noise in the patient's room. When I saw her, soon afterward, there were rhythmical contractions of the left side of the body. The face and neck were in constant motion, and drawn considerably to the left side. The contractions of the face, neck, arm, and leg were synchronous, recurring at regular intervals. The force of the contractions was often irregular in certain groups of muscles. Sensation and consciousness were not affected. She was able to take considerable nourishment. The tongue was protruded with difficulty and slightly drawn to the left side. The right side of the body could be moved at will, but articulation was impossible. The bowels and bladder were emptied involuntarily. Both pupils were contracted, and there developed an active conjunctivitis on the third day in the left eye. The pulse was feeble and rapid, respiration undisturbed, temperature slightly elevated. The movements continued when the patient was asleep with as much force as when awake. The power of the contractions gradually diminished until they were lost in the leg on the fourth day and in the arm on the fifth. She died six days after the attack in an unconscious but not comatose condition.

Autopsy, fifty hours after death.—The body was thin and badly nourished. Face slightly drawn to left side; eyelids of same side swollen and conjunctiva congested, with a muco-purulent discharge. The meninges were healthy; there was some serous effusion at base of brain, but not enough to increase materially intracranial pressure. Upon removal of dura mater a bulging was found in the region of the right parietal lobe. While attempting to separate the brain from its attachments at the base, a clot the size of a hen's egg dropped from the parietal region, the covering of gray matter not being sufficient to support it. The cavity which contained the clot was very superficial, extending in front to the ascending parietal convolution, above to the convolution of the longitudinal fissure, back to near the occipito-parietal fissure, and below to the temporo-sphenoidal lobe. The convolutions pressed upon were the ascending parietal convolution of the longitudinal fissure, the supramarginal, and angular. The ventricles were not disturbed, though there may have been some pressure upon them. The cortical nature of the hemorrhage doubtlessly explains the motor disturbances of the left side of the body. *April* 8, 1886.

6. *Tubercle of left corpus striatum with tubercular infiltration of lungs.*

Exhibited by Dr. H. M. Fisher.

Otto O., æt. about 25 years, a Scandinavian, was admitted to the Episcopal Hospital, March 5, 1886. Upon admission his mind was very dull and he could give no account of his illness. He seemed, however, to be suffering from severe headache. His breathing was somewhat hurried, but he had no cough, and was not emaciated. As he was very rigid and it was difficult to keep him in a sitting posture I neglected to make a careful examination of his chest posteriorly, but over the anterior portions of the lungs detected nothing but harshness of respiration. Ordered potass. iodid. grs. xx every four hours.

March 6. Pronounced stupor. Both pupils react to light. Muscular rigidity more marked. Muscles of both upper and lower extremities respond to stimulation and no paralysis of any muscle or groups of muscles observed. There was, however, complete incontinence of urine. Some twitching of the muscles of the left arm. Difficulty of deglutition. Ordered one ounce of blood to be extracted by cupping over each mastoid process, and one ounce of mercurial ointment by inunction daily.

7*th.* Apparently some return of consciousness; deglutition easier, but excessive muscular rigidity continues, particularly of the dorsal muscles.

8*th.* Ophthalmoscopic examination made to-day by Dr. A. G. Heyl, who reports as follows: Eyelids spasmodically closed, pupils react swiftly to light to barely two millimetres in diameter. In each eye the disk margin is obscured, except the temporal edge, which, especially in the left eye, is quite distinct. The retinal vessels are normal. The appearances at present are not those of choked disk, nor of descending neuritis. The appearances are indicative of intracranial disease, not, however, of meningitis of the convexity, perhaps, also, not of tumor.

10*th.* Marked dyspnœa. Sonorous and sibilant râles audible over pulmonary apices. Pulse irregular; face livid; coma profound.

11*th.* First sound of the heart extremely weak; died in the evening.

Post-mortem examination showed very extensive caseous infiltration of both lungs, only their anterior borders being measurably free from disease. There were several spots of softening and in the right lung a small cavity had formed. No miliary granulations.

Brain: Pia mater showed increased vascularity and at the base of the brain, on the right side, marked thickening. The white substance of

the left hemisphere was much softened, the softening increasing in the vicinity of the basal ganglia. In the substance of the left corpus striatum, a hard, irregular nodule, more or less ovoidal in shape, three-quarters of an inch in length by one-half an inch in width, was found imbedded. Microscopically, this was found to consist of an accumulation of small round cells, giant cells, and some embryonic connective tissue cells. Some vascular loops were found extending only as far as the periphery of these cell accumulations, their walls much thickened. The centre of these cell masses showed granular degeneration and *débris*. The mass presented, in fact, all the appearances of an accumulation of tubercular granulations. *May* 13, 1886.

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7. *Case of cerebro-spinal fever.*

Exhibited by Dr. J. Tyson.

Solomon S., æt. 15 years, was brought to the Philadelphia Hospital near midnight on May 11, 1886. He was in a condition of active, almost maniacal delirium, and it was absolutely impossible to arouse his intellect in any way. The previous history was brief. He had been employed at heavy work for a boy—in shovelling coal and firing an engine. He was said to have been perfectly well up to twelve hours before he was brought to the hospital, but the fact that he had not been working for several hours previous to the attack may be regarded as suggestive of some preceding indisposition or bad feeling. At noon of the 11th he was seen by his employer, and said to be "as well as usual." A half hour or so afterward he was taken sick with an attack of vomiting, and fell into a state of delirium, in which he was oblivious to everything around him. He was taken to his employer's residence, and, after an unsuccessful attempt to have him admitted into the German Hospital, conveyed to the Philadelphia Hospital.

The appearance of the patient at the time of admission was that of a fairly well-nourished, hearty boy. The face had an expression of great fright, due to a marked protrusion of the eyeballs and wide separation of the lids. It was with difficulty that the patient's clothing was removed, for he fought vigorously against any handling, and complained bitterly of his head. Pressure anywhere and any movement seemed to give him great pain, but especially any movement of the head. His frequent cry was, "Oh, my head; oh, my head."

Once in bed he plunged his head into the pillow, drew his knees well up toward his chest, and for a short time fell in a dull, stupid condition with closed eyes and lateral decubitus, from which, however, he was at once aroused by the slightest touch or movement of either extremity. Pulse full and tense, 88. Nothing unnatural about the respiration. Temperature 104$\frac{1}{5}$°. Skin hot and dry. Examination showed an extreme hyperæsthesia, most marked about the head and trunk, but present to a marked degree, also, in the extremities.

There was a tonic contraction of all the muscles of the body, which was readily overcome, and the patient had free use of the muscles of both sides. There was considerable retraction of the abdomen, but no opisthotonos or retraction of the head at time of admission. The pupils were somewhat contracted; there was slight inequality.

After a brief period of quiet and apparently deep sleep the patient became very restless again, tossing about the bed; springing up on his hands and knees, and even upon his feet. His eyes were wide open, and staring frightfully. At the same time he uttered the most pitiful cries.

The following morning there were marked retraction of the head and a slight degree of opisthotonos. A large amount of urine was passed following upon the use of a rectal enema; there had been no retention. The tongue became white coated; the lips dry and brown, and the teeth brown and dirty. The temperature, after reaching 104$\frac{4}{5}$° at 3 A.M., and remaining at this point until 8 A.M., began to fall, until at 2.30 P.M. it was 100$\frac{1}{5}$°. An ice-cap had been constantly on the head.

The patient gradually sank until he died at 7.30 P.M. of failure of respiration. The heart acted strongly but rapidly to the very last.

There was no photophobia; no abnormal markings of any kind on the body; no vomiting after admission. During the last few hours there was a constant trismus.

The irritable state was controlled by a single hypodermic injection of one-fourth of a grain of morphia.

The autopsy showed meningitis of the base of the brain and of the spinal cord. *May* 13, 1886.

8. *Macroscopic lesion in fourth ventricle of a patient dead of diabetes mellitus.*

Exhibited by Dr. F. A. Packard.

Harriet H., white, æt. 52 years, was admitted to Dr. Ashhurst's ward in the University Hospital, suffering from extensive and rapidly extending cellulitis of the hand and forearm. While in the hospital she was, during almost the entire time, in a state bordering on delirium, on which account no satisfactory history of her illness could be obtained, except from her friends. She had been for some time troubled with polyuria, and had had numerous boils in various parts of her body. The trouble for which she was admitted appears to have begun in a palmar abscess which rapidly extended. She, as I have said, presented a peculiar delirious condition, her tongue was dry and fissured, and the skin dry; she suffered greatly from thirst, and passed large quantities of pale urine, which, on examination, showed a large quantity of sugar present. Free openings into the forearm gave marked relief, but did not prevent entirely the spread of the local trouble. She grew continually weaker, until during the latter part of the night before her death she became comatose, and remained so until 6 A. M., when she died.

The following were the conditions found at the post-mortem examination: Rigor mortis but slightly developed. Body well-nourished and with a considerable amount of subcutaneous fat. Skin very dry and shrivelled. On the mons veneris there was a small scab, dried, and apparently the result of a small abscess.

Abdomen and thorax: Layer of subcutaneous adipose tissue thick. Muscles composing abdominal and thoracic walls pale in color. Cartilages of ribs fatty, except that of the first rib, which was ossified. Descending colon situated more anteriorly than usual. Liver large. Other abdominal viscera normal in position. Viscera of thorax in normal position.

Pleuræ: No adhesions present on either side.

Pericardium contained a small amount of clear fluid.

Right lung crepitant throughout. On the inner surface of the lower lobe there was a hard calcareous nodule of about the size of a hempseed. Whole lung deeply pigmented.

Left lung crepitant throughout and deeply pigmented. On the inner surface there were to be felt and seen numerous small, rough, calcareous points.

Heart large. Right auricle contained a large, dark clot extending into the vena cava; when the clot was removed there was a great flow of dark liquid blood from the cava. Right ventricle contained a large two-layered clot extending into the pulmonary artery. Left auricle was empty. Left ventricle contained a few pale clots closely connected with the papillary muscles. Externally, the heart was coated with a large, thick layer of fat; walls were of normal thickness. Right side of heart normal as to valves. Mitral valve was thickened at edges of leaflets and hardened. Aortic valve showed slight calcification.

Coronary arteries not calcified.

Aorta atheromatous in numerous patches.

Stomach was normal in size, and contained grayish semi-digested food of sour odor. Mucous membrane presented numerous small ecchymotic points. Pyloric orifice normal.

Duodenum showed slight congestion, and contained some semi-digested food.

Bile-duct patulous.

Intestines normal in color externally. Mucous membrane of upper portion of small bowel much congested. In lower portion of small bowel there were numerous streaks of coagulated blood adherent to the mucous membrane. Peyer's glands healthy in appearance. Sigmoid flexure very long and tortuous. Vermiform appendix long and contained greenish feces. Mucous membrane of large bowel appeared healthy.

Spleen large and soft. Pulp soft and dark in color. A white patch existed on its anterior border.

Liver large, extending completely across the abdominal cavity; redder than normal and soft. Portal and hepatic veins free. Gall-bladder small and contained a small quantity of bile. Gastro-hepatic omentum thick and tough.

Peritoneum was everywhere thick, tough, and full of fat; it formed a complete meso-colon.

Pancreas appeared normal.

Suprarenal body: No suprarenal body could be found on the right side, although careful search was made for it. On the left side the suprarenal was at least three times as large as normal and of a dark color.

Kidneys: Right kidney large and pale; capsule stripped off readily, except at one point at the upper posterior portion, where some of the kidney substance was torn away with it. On section, it was pale, small vessels plainly visible, and small arteries showed cut-ends plainly; Malpighian tufts prominent as dark points. Left kidney slightly larger

than normal and pale; on section presented the same appearances as the right.

Bladder distended. On an opening being made there flowed out a quantity of grayish urine with a sweetish odor.

Uterus of normal size. A plug of glairy mucus filled up the os. On section, the veins of the cervix were found to be congested. Mucous membrane slightly thickened. From the extreme fundus there projected into the cavity a non-pedunculated growth of about one inch in length aud two-thirds of an inch in breadth, firm, and covered by mucous membrane.

Fallopian tubes somewhat narrow, otherwise normal.

Ovaries small, extremely bloodless, and puckered with cicatrices.

Head: On dividing the tissues of the scalp a large quantity of dark venous blood escaped. Skull-cap of normal thickness; dura mater very adherent. Longitudinal sinuses distended with dark liquid blood. Pacchionian bodies numerous. Dura mater slightly thickened. Lateral and occipital sinuses filled with dark fluid blood.

Brain was small. Over the surface there was an œdematous appearance. Vessels of pia mater congested; vessels at base normal. Punctæ vasculosæ few and inconspicuous. In each lateral ventricle there was about a drachm of clear serum. On the right side the choroid plexus of the fourth ventricle was abnormally adherent to the roof and lateral wall of the ventricle, and there were three or four slender adhesions extending between the plexus and the floor. *May* 27, 1886

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9. *Tubercular tumors of the cerebellum.*

Exhibited by Dr. G. E. de Schweinitz.

Lizzie D., æt. 4 years, was admitted to the Surgical Ward of the Children's Hospital March 17, 1886, for arthritis of the right elbow, and the effects of apparent neglect and poor food. Her father is living, and would be a healthy man except for occasional attacks of rheumatism. The mother is also alive, and has been a hard drinker; six months ago she became insane. Of the six children which composed her family, the eldest is 15 years, but her withered and prematurely aged face makes her appear many years older. One brother died of Pott's disease. This child, the youngest, has always been feeble, and easily "took cold." Eighteen months ago she sustained an injury to the right elbow, which

resulted in arthritis. Eight weeks before admission she began to lose the power of her lower limbs, and when admitted was unable to walk. After admission no other symptoms of note were recorded, except a peculiar staring expression of the eyes and apparent indifference to surrounding objects. My attention was called to this, and in making an ophthalmoscopic examination I found a complete picture of subsiding optic neuritis. The appetite, digestive functions, and mental faculties were normal. An examination of the urine showed the presence of triple phosphates. For a time the case remained stationary, but about the beginning of June she began to fail rapidly. On June 17th vomiting and obstinate constipation were marked symptoms, and June 21st convulsions set in, and were more or less constant until death. On the evening of June 22d the breathing assumed the character usually described as Cheyne-Stokes respiration, and about 4 o'clock on the afternoon of June 23d the child died. The house-surgeon, Dr. Kerlin, made the post-mortem examination in the presence of Drs. Wharton, Lewis, Martin, Randall, and myself, about twenty hours after death.

Autopsy.—No unusual external appearances.

Thorax: The right lung, pleural cavity, heart, and pericardium showed no abnormalities. On the left side there were extensive pleuritic adhesions from apex to base, and the upper lobe of the lung was small, shrunken, completely bound down, and on section cut like a fibrous mass.

Abdomen: Position of viscera normal, and, except for some enlargement of the mesenteric glands, there were no lesions discoverable.

Brain: Dura mater normal, except for a few slight adhesions along the longitudinal fissure. The anterior portion of the brain was flattened. and there was some subarachnoid effusion. Both lateral ventricles, and also the third ventricle, were distended with a large quantity of clear fluid; otherwise no abnormalities of the cerebrum were present. At the base of the brain there was some opacity of the membranes, but no lymph; on the upper surface of the right cerebellar hemisphere the dura was tightly attached, and beneath incorporated with the tissue there was a dense, yellowish-white mass; on the left hemisphere a similar attachment was observable, though smaller and hard. Both optic nerve sheaths showed well-marked ampulla behind the globe.

Later microscopical examination revealed the following points: The tumors were solitary tuberculous masses undergoing caseation; tubercle bacilli were found in the larger. The optic nerve showed in its whole length descending neuritis with beginning atrophy. The pia mater of the base was affected with beginning tubercular meningitis; the upper

lobe of the left lung was converted into fibrous tissue and numerous small round cells, probably a tuberculous process undergoing fibroid change. *June* 24, 1886.

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10. *Miliary aneurisms of the brain.*

Exhibited by Dr. J. P. C. Griffith.

I beg leave to report the following case of hemiplegia, which had been under my care for some time, and which recently came to the *sectio cadaveris:*

Mrs. F. was a lady of about 70 years of age, with paralysis of the right side of the body; greatly increased patellar reflex, ankle clonus, and some contraction of the tendons. On September 4, 1886, she exhibited vomiting, stertor, total unconsciousness, congested face, and a full, slow pulse of very high tension. In less than twenty-four hours she was dead. At the autopsy the veins and sinuses of the brain were found full of blood; and blood oozed at the base from the infundibulum. Both lateral ventricles contained clotted blood, and a small clot joining the masses on each side filled the foramen of Monro. The third and fourth ventricles also contained blood. The left caudate nucleus and the anterior part of the lenticular nucleus were entirely disorganized; the optic thalamus uninjured. An old scar was found at the posterior part of the left lenticular nucleus, between it and the optic thalamus, and largely within the internal capsule. Upon careful dissection and washing away of blood, and upon gentle traction of the bloodvessels of the anterior perforated space on the left side, two or three small aneurisms, the size of a split pea, were discovered, and a similar one was found on the right side. I present these for the inspection of the Society this evening. It is evidently from the rupture of some such aneurism that the hemorrhage came. A more careful search revealed a miliary aneurism of one of the smallest vessels of the anterior perforated space. This may be seen under the microscope on the table. No doubt there were many others undetected. *September* 23, 1886.

11. *Sarcoma of the left frontal lobe.*

Exhibited by Dr. W. Osler.

Unknown man, admitted December 8th. He was unconscious and thought to be drunk, and was placed in the ward for inebriates. He had slight fever, but no local disease could be detected. Death took place on the third day.

Brain: Slight turbidity of vessels at base. Under surface of frontal lobes looked smooth and full, particularly noticeable near chiasm. Immediately above the left olfactory bulb the pia was swollen and congested. The brain substance beneath it for an area equal in size to a quarter dollar, was ecchymosed. Vessels of circle of Willis normal. On separating the longitudinal sinus the membranes were infiltrated and œdematous; no extravasation. The arteries, large and small, at the base were unusually distended with blood. On the surface of the brain the veins were very full. The small vessels over convolutions were very prominent. The left frontal lobe was swollen. In the frontal section, by Pitres' method, there was exposed a tumor the size of a walnut, occupying the anterior extremity of the frontal lobe, extending close to the apex; at its lower part it was cystic and contained a yellowish fluid. The second section, cut at a distance of two and a half inches from the fissure of Rolando, exposed an infiltrated yellow tissue; the white matter in the neighborhood was much swollen and œdematous. The third section, immediately through the base of the frontal convolutions, exposed a normal white matter. This section cut through the anterior extremity of the corpus striatum. Section through ascending frontal convolution showed no change. Section of the optic nerve near the eye, showed the sheaths swollen; no neuritis. Examination showed the tumor to be a gliosarcoma, with cells of larger size than usual. *January* 27, 1887.

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12. (1) *Tumor of the second frontal gyre.* (2) *Tumor of the optic thalamus. Remarks on the localization of oculo-motor and facial centres.*

Exhibited by Dr. C. K. Mills.

The specimens herewith presented are of unusual interest from several points of view. If the symptoms exhibited by the patient from whom the first specimen was taken were due to the only lesion which I was

able to find, the case is unique, as it shows oculo-motor paresis and upper facial paralysis apparently due to a cortical lesion, or to a lesion conjointly of the frontal cortex and centrum ovale. The patient from whom the second specimen was removed, had also marked paralysis in the upper distribution of the facial nerve as well as the lower, and this was associated with hemiplegia and some hemianæsthesia of the same side.

W. V., æt. 16 years, male, was admitted to the Philadelphia Hospital about two weeks before his death. His mother died of heart disease; his father is living, and is a hard drinker. He had two sisters and one brother, who are healthy; one brother, however, died in infancy of hydrocephalus. He had measles and scarlet fever about eight years ago, and dropsy followed the latter. He had been compelled to work beyond his strength for years. He had had occasional convulsions for over a year, and thought that he had received a blow on the head before they came on. He had a scar about an inch long in the scalp on the right side of the head, but it seemed to be superficial; no depression or cleavage of bone could be made out.

On or about December 27, 1886,[1] in trying to rise from his chair, he fell. He had noises in his head, or, as he expressed it, "his head moaned." He also had vomiting at this time, and obstinate constipation; and sound and light intensified the pain in his head. He had some fever and his pulse was sometimes as low as fifty to the minute. The next day there was some loss of power and he walked with a staggering gait; he had headache and cold sweats. On the third day he had diplopia, vertigo, and palpitation; and he now cried out with severe pain in his head, and could retain nothing on his stomach. He also had seizures, the nature of which could not be determined. These got somewhat better, but were followed by sensations of pins and needles in the arms, accompanied with great stiffness. His bowels continued to be much constipated. In the second week of his illness he lost power to some extent in his left arm, and had some depression of sensation in the right. Although during this week he seemed to be stronger, on attempting to walk he staggered and fell. He had intolerance of light all the time, especially marked in the right eye. During his fits he was drawn to the left side, and always cried out before they came on. He had sensations of choking during the fits, and said that he was never unconscious in them. He was ravenously hungry most of the time during his illness after the first week, and bolted his food at all times.

[1] During the early part of his illness he was attended by Dr. E. H. Speer, who kindly sent me some notes of his case.

His temperature on admission was 99°, but afterward ranged between 98° and 99°. He had attacks of vomiting at irregular periods, and his bowels were much constipated but readily moved by enemata. His eyes were somewhat immobile or slow to move, making his gaze seem somewhat fixed. His pupils were equal. He had a slight but distinct ptosis of the right eye, sometimes more marked than at other times. He could not close the left eye nor wrinkle the left forehead (paralysis of the upper fibre of the facial nerve). With the dynamometer the right hand marked 15, and the left 9. He had distinct loss of power in the left arm and hand, and also in the left leg, but not so marked. He died apparently from general exhaustion.

The following are the notes of the autopsy: The body was that of an emaciated boy; there were several scars on the head. Slight adhesions of pleura were found in the upper portion of the right lung; the lungs were otherwise normal. The heart, kidneys, and liver were normal. The intestines and spleen were not examined.

The dura mater was not adherent to the skull, but on attempting to remove it the right side was found to be adherent strongly to the pia mater and the brain underneath it over the antero-frontal region of the brain, and the corresponding portion of the median surface of the right hemisphere. In removing the brain a little serous or grumous fluid exuded from this region, but no true pus. On carefully removing the dura a reddish-gray grumous mass was found adherent to the agglutinated dura and pia mater, and involving the gray matter of the brain and a portion of the white matter immediately adjacent. This was soft and in its centre a hole or cyst appeared, or the space had been occupied by tissue now broken down. The exact location and dimensions of this mass were as follows: it was almost entirely confined to the posterior portion of the second frontal gyre (of Ecker), encroaching very slightly at its posterior and lower part upon the lower third of the ascending frontal gyre. Its greatest dimension was diagonally from below upward and forward, three and a half inches. Perpendicularly the broken-down mass extended two and a half inches, and also two and a half inches antero-posteriorly. It involved the white matter of the second frontal gyre, and to some extent that of the first and adjacent portion of the third. The ganglia and capsules proper were not involved. There were some meningeal congestion and inflammation continuous with the mass.

It is interesting to note that in this case a mistake in diagnosis was made. The existence of ptosis of one side, and of paralysis of the upper fibres of the facial nerve of the other, with paresis of the arm and leg of this side, led me to infer that the case was one of more or less diffused

meningitis with exudation at the base. It looked as if the facial and oculomotor nerves of opposite sides, at their superficial origins, had been more or less involved in some process causing pressure to be exerted upon them, and that the descending motor tracts were so affected as to produce paresis of arm and leg. The most careful scrutiny of the base, examining one nerve after another at its origin, and as far as possible along its course, revealed nothing, and unless the microscope should show some pathological condition which I have not been able to discover with the naked eye it is reasonable to infer that the symptoms present were due to the only lesion discovered, namely the tumor involving the posterior part of the second frontal gyre, and a small portion of the ascending frontal gyre, and the adjacent white matter. It is not my purpose to go into any lengthy comments upon the case, but rather to submit it as a clinico-pathological contribution to the study of localization to be used by others or myself at some future time. It is unfortunate that no ophthalmoscopic examination was made in this case. Arrangements were made to examine the eyes carefully with the ophthalmoscope, but through a combination of unfortunate circumstances it was not done.

According to Ferrier,[1] at the base of the first frontal and extending partly into the second frontal gyre in the brain of the monkey is an area, irritation of which causes elevation of the eyelids, dilatation of the pupils, conjugate deviation of the eyes, and turning of the head to the opposite side. If the stimulation is faint, elevation of the eyelids is the only observable effect. The same authority says that certain clinical facts seem to require the existence of a distinct centre for the levator palpebræ superioris, inasmuch as paralysis may occur limited to this muscle without affecting the other muscles supplied by the third nerve. Ferrier opposes the view of Landouzy that the centre for the levator palpebræ superioris is situated in the angular gyre, holding that as ptosis does not necessarily accompany lesion of the angular gyre, no causal relationship can be considered as established between the two. He holds that if the distinct volitional centre for the levator palpebræ superioris is in the human brain, it must be sought for in the region constituted by the bases of the first and second frontal gyres.

"There are, however," he says, "difficulties in the way, for lesions of this region are not uncommon, whereas isolated affection of the levator palpebræ is a comparatively rare occurrence, or at least has not been so obvious as to call for remark. This absence of paresis or paralysis, there-

[1] Localization of Cerebral Disease, p. 38.

fore, requires explanation. It seems to me not improbable that the intimate bilateral association of the oculo-motor nuclei may account for this, and that the escape of the levator palpebræ from paralysis in cases of unilateral lesion is simply a higher degree of that which is seen in the orbicularis oculi in facial paralysis of cerebral origin."

It will be noticed in this case that the ptosis was on the same side as the lesion. If not conjugate deviation, there was, at least, some immobility of the eyes. Diplopia also was present for a time. The patient seemed sometimes to have slight right internal squint.

D. M., æt. 19, white, was born in Philadelphia. His father died of bronchitis at the age of 39. He was wild and wayward, not living at home. He was bitten by a horse in the hand during the latter part of June, 1886, and was paralyzed about a week after he was bitten. He was sweeping a store when he suddenly fell, and on rising, in about five minutes found his right side somewhat paralyzed. He complained of much tenderness over the abdomen. He did not suffer with headache before, but his head ached persistently after this time, the pain being most marked over the left eye and round the left temple, though it extended more or less over the whole head.

He had a slight cough which was worse in the morning, with some mucopurulent expectoration. He had prolonged expiration with crackling râles at the left apex, with a slight impairment of resonance. His right lung was normal. The heart sounds were nearly normal, although the second was somewhat accentuated, and the first slightly muffled. He suffered from anorexia and frequent vomiting.

When first examined his face was drawn markedly to the left side; he could not wrinkle the right forehead, and there was lagophthalmos of this side. He could not raise his right arm to a level with his shoulder, and was unable to walk well because of paralysis of the right leg. Later, the hemiplegia became profound. He had a blotchy, irregular, erythematous and papular eruption over his chest. His hair had fallen out considerably. The æsthesiometer showed some loss of sensation in the right limbs.

Jan. 18, 1887. His pupils were irregular, the left contracted. He was very dull in the morning and had delusions. His headache was not so severe as before.

19*th.* He was considerably brighter. Iodoform ointment was rubbed over the nape of the neck, and three grains given internally. Some improvement was noted in the morning. The external rectus of the right eye was paralyzed. His eyes were bloodshot and his tongue coated. He had no delirium and he took nourishment well.

20*th.* His pulse was very rapid, about 160. He complained of much pain in the back of his neck, especially if he moved. He had some difficulty in swallowing. He died on the 21st. From January 11th to January 21st, his temperature most of the time ranged between 97.5° and 100.5°, and oscillated irregularly. A complete record of it was kept.

Autopsy.—(Made by Dr. Wm. Osler.) His body was that of a poorly nourished lad. There were adhesions of the left pleura. The heart was of average size; on the right side the tricuspid ring was wide and would admit three fingers and a thumb. On the left side the mitral valve was normal, and the aortic was much fenestrated; the fenestrations were large; there were numerous small vegetations. The aorta was smooth. The lungs had a cavity at the left apex. Throughout the upper lobe were numerous caseous masses. In the lower lobe were numerous nodular groups of tubercles. The right lung was crepitant throughout, and presented numerous scattered groups of tubercles. The bronchial glands were slightly enlarged, containing caseous and calcareous masses. The abdominal organs were normal. The dura mater was normal. At the base of the brain the membranes were clear. The vessels over the convolutions were clear. A little thickening and matting of the membranes were noticeable in the Sylvian fissure, and on full exposure there were seen in the pia, covering the convolutions of the island, numerous gray granulations. The ventricles were found much dilated. The amount of fluid was excessive. The septum and fornix were almost diffluent. The posterior cornu was especially dilated. On the left side the thalamus was occupied by a large mass, which projected on the floor of the ventricle, covered by the velum, which was thin and stretched over it. The mass seemed to project from the inner portions of the left thalamus and pushed away the outer part and the caudate nucleus. The mass was grayish-red in color, very regular on the surface, and very firm. It projected forward to within a short distance of the head of the caudate nucleus. A section through the anterior portion of the tumor showed that it had pushed aside the thalamus, and it appeared to occupy a portion of its inner half. The gray matter in its vicinity, as well as the internal capsule, was infiltrated and œdematous. On section of the left hemisphere the ganglia seemed normal. On further examination a few tubercles were found in the anterior perforated space.

One point of interest in connection with this case is the same as one presented by the first case, namely, the existence of complete facial paralysis in association with hemiplegia. The muscles in the distribution of the upper fibres of the facial nerve, namely, the frontalis, orbicu-

laris palpebrarum, etc., were profoundly paralyzed. Commonly, as is well known, in hemiplegia from cerebral lesions—from lesion anywhere above the pons—the upper face escapes, or, if paralyzed at first, promptly regains power. Out of the multitude of hemiplegics, therefore, we have few instances of upper facial paralysis associated with helplessness of the arm and leg of the same side, although the lower portion of the face is usually affected to a greater or less extent. I have, however, seen several cases in which paralysis of the arm, leg, and the entire face on the same side were present. Some of those cases are on record, a few were collected by Dr. Lloyd and myself, and are to be found in the article on "Tumors of the Brain and its Envelopes," in the *System of Practical Medicine by American Authors*, edited by Dr. Wm. Pepper.

The following remarks occur in a paper presented by me to this Society January 23, 1879 (in this paper was described a case of tumor of the brain situated just in front of the optic chiasm): "A few cases are on record, for instance, in which the upper fibres of the facial nerve have been paralyzed, as the result of a lesion situated in the brain, above the pons and cerebral peduncles. Chvostek, quoted by Nothnagel (Ziemssen's *Cyclopedia*, vol. xii. p. 118), describes a case in which disturbances of speech and a feebleness of the extremities of the left side, present at first, disappeared almost entirely after a few days; on the other hand, the left facial nerve remained almost completely paralyzed (even the branch to the orbicularis palpebrarum), and the autopsy disclosed the presence of a hemorrhagic cyst in the right nucleus lenticularis. The following explanation of the cases of this kind is given by Nothnagel (*op. cit.*, p. 147):

"'When the tract of the nerve fibres passing along the base of the nucleus lenticularis, designated as the *ansa peduncularis* (Hirnschenkelschlinge, Gratiolet, Meynert), is involved in the lesion, the character of the symptoms is somewhat different from that described. The most noticeable difference seems to be that in this case the fibres of the facial nerve supplying the frontalis and the orbicularis palpebrarum, which otherwise escape, are paralyzed with the rest, as in the instance reported by Huguenin.'"

In the present case the upper facial paralysis is probably to be explained, not from the situation of the tumor in the thalamus, but either from the pressure exerted by it, or the inflammation and œdema adjacent to it, or from both conjointly. In this indirect way both the capsule and lenticular nucleus were involved.

The paralysis of the upper face in the first case must be explained differently. If due to the cortical lesion, the tumor or cyst probably

destroyed either the centres for the movements of the eye and forehead, or the white matter constituting the tracts which connect the centres with the parts below. It will be remembered that the lower third of the anterior central or ascending frontal gyre was infringed upon, and that the destruction of white matter extended somewhat beyond the second frontal gyre.

It has occurred to me that the persistence of both the ptosis of one side, and the paralysis in the upper distribution of the facial nerve on the other side, may be explained each from the other. It is supposed that upper facial paralysis and oculo-motor paralysis of cortical origin do not persist, as already stated in quoting Ferrier, because of the intimate bilateral association of the oculo-motor and of the facial nuclei. In this case, supposing the ptosis to have been due to the cortical lesion on the same side, and the facial paralysis of the other side to the same lesion, a curious disassociation was produced, and hence the unilateral symptom in each case persisted. *February* 24, 1887.

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13. *Glioma of the right temporal lobe, with intercurrent hemorrhage. A case in which the question of trephining was considered and decided against.*

Exhibited by Dr. C. K. Mills and Dr. G. A. Bodamer.

The chief symptoms and conditions were as follows: severe headache, more localized in right temporo-frontal region; pain on localized pressure and percussion; impairment of sight and hearing; choked disk; dilatation of right pupil; three days before death paralysis of left arm and paresis of left leg, paresis of right face, ataxic aphasia.

H. B., æt. 12 years, was admitted to the German Hospital, April 12, 1887. The father of the patient died suddenly from the effects of a sunstroke; the mother from cholera morbus. The patient was the youngest of six children, and the only survivor, the rest having died in infancy, childhood, or youth, but from what diseases could not be ascertained. The patient was well until she was two years of age, when, according to the statements furnished by a relative, she became wholly paralyzed, and remained so for four weeks, after this slowly ceasing to be helpless, although her gait always continued to be somewhat uncertain and peculiar. She was sickly for two to three years, when she

regained her general health completely. She was sent to school, but was rather dull.

Toward the end of September, 1886, she fell from a front door-step, a height of four feet, and was picked up unconscious, in which condition she remained for about ten minutes. In December, 1886, and January, 1887, she suffered from suppurative inflammation of the ear, and, at times, from nose bleeding. After the fall she always complained of headache, which became very intense early in April, 1887, and so continued until the time of her death. When admitted to the hospital, April 12th, her chief complaint was of this headache, which she localized especially over the right temporo-frontal region. Some tenderness to pressure and percussion was present over this same region. Her tongue was thickly coated, but she had no fever or general symptoms. Examination shortly after admission showed both pupils somewhat dilated, the right, however, undoubtedly larger than the left. She had some difficulty in seeing, and her hearing was somewhat defective, or, at least, it was difficult, at times, to fix her attention, or get responses without loud questioning. Unfortunately, no detailed study of sight, hearing, or the other senses, was made. At the time of admission to the hospital she gave no evidences of paralysis, or of any other symptoms than those above detailed.

Until the evening of Friday, April 29th, her condition continued as above described. At this time she suddenly became paralyzed in the left arm, and paretic in the left leg; her speech was also affected, articulation becoming for a time so indistinct and imperfect, that nothing she attempted to say could be understood. She did not become completely unconscious at the time of the paralytic attack; she did not have either Cheyne-Stokes or stertorous breathing. Her mouth was slightly drawn to the left, her pupils remaining dilated, the right continuing larger than the left. So far as could be determined sensation was not affected on either side. Ophthalmoscopic examination showed choked disk in the right eye. The condition in the left eye was not positively determined, although the vessels were engorged.

On May 1st a consultation was held to consider the question of trephining, for the removal of the supposed growth. At the consultation were present Drs. Adam Trau, D. Hayes Agnew, F. H. Gross, J. B. Deaver, and C. K. Mills, with the resident physician of the hospital, Dr. George A. Bodamer. After some discussion, it was concluded best not to trephine. This conclusion was reached because of the presence of certain symptoms and the absence of others, which threw doubt upon the exact location of the growth. Apparently, at first sight, the case

was one of brachial or brachio-crural monoplegia, from lesion of the arm centres of the cortex and adjacent areas. The paralysis of the arm and paresis of the leg were the most marked conditions. The patient, however, had also dilatation of the right pupil, and either paresis of the lower right face, or slight spasm of the lower left face. It was really somewhat difficult to determine whether the condition was one of paresis on one side, or very slight spasm on the other. The left corner of the mouth was slightly drawn up, the right apparently drooping somewhat. It was argued that if the condition was one of right-sided facial paresis, the paralysis of the limbs associated with this condition could not be explained by a single growth of the cortical centres of the right side; neither was the dilatation of the right pupil easily explicable from the standpoint of a lesion of the arm and leg centres of the cortex. The entire absence of spasm, either local or general, was also somewhat against the probability of the tumor being one so superficially localized in the motor zone, and connected with the membranes as to be readily reached by operation. Finally, the sudden occurrence of marked paralysis, after the long continuance of other symptoms of brain tumor or abscess, showed that either a hemorrhage had occurred, as was suggested by Dr. Trau, or that a sudden and somewhat extensive break-down of tissues in the vicinity of the old lesion had taken place. The suddenness of the paralysis seemed to negative the idea that it was the result of the extension of the growth by slow development from a latent to the motor region, although this idea was considered. On the whole, the conditions seemed to point to a growth either at the base on the right side, or so low down in the cerebrum as to exert pressure upon the descending motor tracts and, possibly, also upon some fibres of the facial and oculomotor nerves of the same side. The conditions as to consciousness and speech were peculiar. The patient certainly understood much, if not all, that was said to her on May 1st, although the voice had to be somewhat loud and the manner earnest in order to get response. Her speech was thick and indistinct; the aphasia was ataxic rather than amnesic. On asking her what her name was, she answered "Hattie," so that she could be understood, although the articulation of the word was thick and imperfect. The patient died the morning of May 2d.

Autopsy.—At the autopsy, which was held thirty-six hours after death, were present Drs. Adam Trau, F. H. Gross, Charles K. Mills, and George A. Bodamer. The skull was found to be unusually thin, and roughened in a peculiar way on its inner surface; and here and there were jagged processes, especially in the occipital region. A considerable area of the calvarium was injected; and the outer surface of the dura

mater showed corresponding streaks and patches of a bright red color, markedly in the postero-frontal, parietal, and occipital regions. The appearances were present on both sides, but were much more decided on the right. When the dura mater was removed, the exposed pia mater and surface of the brain at first showed nothing abnormal, except here and there a few spots of unusual coloration. Evidently no growth connected with the membranes was present on either the median or lateral aspects of the brain.

The removal of the brain from the skull was conducted with great care, but during the process a slight break through the surface of the brain occurred at a point about the middle of the right second temporal gyre, and through this poured out a mahogany-colored fluid. The tear enlarging, a cavity and growth were revealed. The neoplasm was a frangible, semi-solid, light purplish mass, having beneath and partly around it a cavity containing detritus, and a large unorganized, and evidently recent, clot. The space occupied by the neoplasm, cavity, and clot was irregular in shape, its greatest length being about three inches, and its height and width probably about one-half its length. It occupied a large portion of the interior of the right temporal lobe, but was strictly limited to it. Careful sections showed that the anterior one and one-half inch of the lobe was not involved, and the tissue destroyed did not extend quite to the temporo-occipital junction.

A microscopical examination was made by Dr. Bodamer. This revealed a hemorrhagic or rather a very vascular glioma.

May 12, 1887.

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14. *Large tumor of the right frontal lobe without the presence of active symptoms.*

Exhibited by Dr. G. E. de Schweinitz.

John S., æt. 8 years, was admitted to the Surgical Ward of the Children's Hospital August 31, 1886. The father is living, and apparently a perfectly healthy man. From him no history of syphilis could be obtained. The mother died of phthisis. This boy, with two others, composed the family, one older and one younger, and both healthy children. This patient's illnesses previous to the date of his admission were measles, whooping-cough, and smallpox. Three years ago swelling of the left ankle began, and a few months later of the right elbow. There was no history of injury to account for the joint affections.

His condition on admission was the following: All the superficial lymphatic glands were much enlarged. The right elbow was enormously swollen, tender, and of a dusky red color. Several sinuses opened on the surface and led down to the joint, which was advanced in disease. The left ankle presented the same condition as the elbow. The tarsus was extensively carious, and there was decided acquired talipes varus. The left thumb was swollen, red, and tender, and presented the appearances of syphilitic dactylitis. There was a large

FIG. 1.

Photograph of child, showing the diseased ankle, elbow, and thumb, also the broken-down area below the axilla.

broken-down area below each axilla. The teeth were irregular and the cutting edges of the central incisors markedly serrated. In October, 1886, Dr. John Ashhurst excised the right elbow-joint and amputated the left thumb. The patient soon recovered sufficiently to be out of bed. In March, 1887, Dr. Samuel Ashhurst made a partial resection of the left tarsus. The child did well until May 14th, and never showed any symptoms of brain disease.

On the morning of this date he was unusually irritable, and during the dressing of his foot apparently suffered pain; afterward until 1 o'clock he lay in a stupefied, though not unconscious state. Then, while being helped to a bed-pan, he was seized with a convulsion, which is thus described by the attending nurse. The convulsive movements began on the left side, and were most marked in the face, neck, and arm, with later involvement of the chest and leg muscles. The members of the right side were not quiescent, but were convulsively elevated and drawn toward the median line. The eyes and head were violently turned toward the left side. The convulsion lasted three minutes, and after its cessation the following conditions obtained: There was complete right-sided hemiplegia and contractions of the left side with the production of a typical "claw-hand." Attempted movement of the limbs of this side was resisted in every direction. Neither anæsthesia nor hyperæsthesia existed, though apparently sensation was more acute upon the left side. The pupils were dilated unequally, the left measured seven millimetres and the right five millimetres in the presence of candle-light. There was marked conjugated deviation of the head and eyes to the left. A slight nystagmus was occasionally manifest. The respirations varied; at times they were typically Cheyne-Stokes, at other times deep and almost stertorous, and again, short and gasping. The urine was free from albumin and sugar, and deposited freely triple phosphate crystals. Numerous convulsions succeeded the first one, which was the most severe. They were observed by Dr. Morris J. Lewis, by the house-surgeon, Dr. Harvey Shoemaker, and by myself, as follows:

The convulsions began on the left side with a spasmodic closure of the eyelid and twitching of the face, neck, arm, and leg. The convulsive wave then rapidly passed to the members of the right side, which were in turn convulsed and drawn toward the median line. These convulsions became milder and milder, until before death they consisted simply of twitchings confined to the left side.

On the evening of the day on which these manifestations began I examined the eye-grounds and found double optic neuritis. The edges of the disks were hidden by the swollen retinas, the surfaces of the elevations were gray-white, and there was wide distention of the lymph sheaths. The veins were full and tortuous, and the arteries by comparison small. No hemorrhages were noted. Death took place thirty hours after the first convulsion with no changes in the symptoms detailed, except a gradual failure of the pulse, and several hours before death contraction and equalization of pupils to two millimetres.

Autopsy, six hours after death.—Body of an exceedingly wasted male child. General lymphadenoma; in the groin bunches above and below Poupart's ligament. Submaxillary and axillary glands all enlarged, those in right axilla suppurating. Excision of the right elbow-joint and tarsectomy of the left ankle. Enlarged gland just above the head of the left radius; trochlear glands enlarged.

Thorax: Partly calcified and enlarged lymph glands in the anterior mediastinum under the manubrium. Bronchial glands of the posterior mediastinum very large and cheesy, the entire group of bronchial glands presented this condition and scarcely any trace of gland tissue remained. Those most affected lay immediately anterior to the right bronchus. *Lungs:* A few adhesions at the right apex. Lungs crepitant throughout; at apices a few small nodules, and here and there on the pleural surfaces were scattered fibroid and calcified tubercles. There were *no caseous areas*, except in the right lung, between the middle and upper lobes, a small partly cretaceous and caseous mass. *Pericardium and heart:* Pericardium smooth. Right heart dilated and full of clots, in the left the blood was fluid. All valves normal.

Abdomen: No peritoneal tubercle. *Liver:* Large, firm, the larger vessels distended. No gross changes noted. *Spleen:* Much enlarged, its hilus occupied by four or five caseous lymphatic glands to which was attached the tail of the pancreas. The body of the pancreas was normal. Section showed no tubercles in the pulp of the spleen, which was soft. *Kidneys:* Large, veins full, cortices opaque in places.

Stomach and intestines: Stomach almost empty and mucosa showed no changes. In the ilium several of Peyer's patches swollen and firm, but not distinctly ulcerated. Mesenteric and retroperitoneal glands all enlarged and cheesy.

Brain and membranes: Head large, symmetrical. Skull cap thin. Dura not adherent, smooth and very tense. The convolutions were swollen, sulci narrow. Gray matter not specially congested.

Inspection of the base: The right hemisphere looked larger than the left, particularly the frontal lobe, which, on separation of the longitudinal fissure, projected considerably beyond the median line, depressing the left median convolutions. On the under surface of the right lobe at the anterior part of the inferior frontal convolution there was a grayish white mass, which measured superficially 3 cm. in diameter. The posterior part of the convolution was soft and œdematous, and this condition existed in a less degree throughout the orbital gyri. On separating the operculum a firm, solid mass existed immediately outside the root of the olfactory nerve at a point corresponding to the junction of

the third right with the anterior gyrus of the insula. Further dissection revealed that this mass occupied two or three of the anterior gyri of the insula. The right middle cerebral artery ran over it, and its branches looked smaller than the corresponding ones on the left side.

Fig. 2.

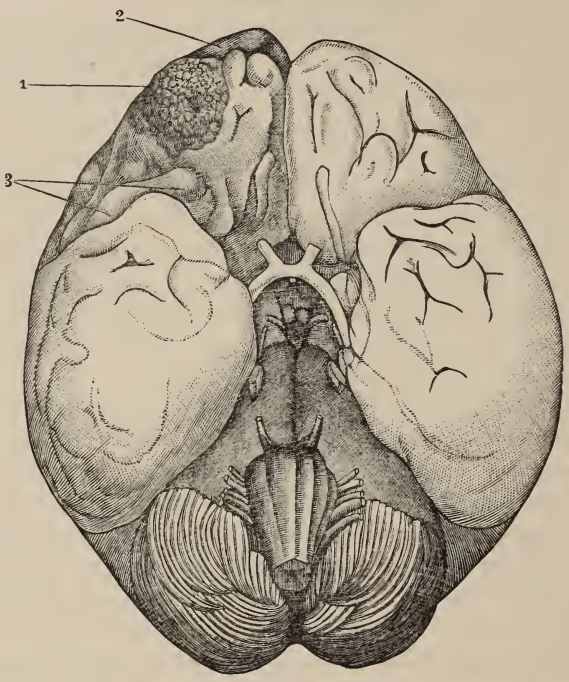

Plate showing base of brain; 1, exposed part of the tumor; 2 and 3, areas of œdematous brain tissue.

There was no special matting of the pia over this mass. The optic nerves were firm and there was wide distention of their sheaths behind the globes. The optic tracts were spread as they passed over the crura. Over the pons and medulla the pia was thickened and infiltrated. The cerebellum was removed by cutting through the crura and was normal. The lower part of the *left crus* was in a condition of hemorrhagic infiltration, and there was a hemorrhagic exudate on the posterior end of the *left thalamus.* The pia, as it dipped in here, was matted and infiltrated. Pons and medulla showed no changes. The ventricles and ependymal veins were distended. In the right lateral ventricle a large grayish white mass was apparent, which occupied the head of the caudate nucleus,

the body of which was œdematous. This œdematous condition extended to the anterior portion of the thalamus. No other lesions were found.

Dissection of the tumor region: After hardening the right half of the brain in alcohol, a careful dissection of the tumor region was made. The tumor extended from about half an inch of the tip of the frontal lobe to a point corresponding to a section through the bases of the first and second frontal convolutions, about one inch anterior to the fissure of Rolando. Anteriorly it occupied the greater part of the white matter of the lobe, and came to the surface at the junction of the third frontal with the orbital gyri. Its depth was about two and a third inches. It consisted here of two tumors, the larger below, and the smaller above, separated by soft brain tissue. As it passed back it reached the orbital surface, and was apparent immediately behind and external to the root of the olfactory nerve. Its posterior limit was three and a half inches from the tip of the lobe. Section through the prefrontal sulcus showed that here the thalamus and caudate and lenticular nuclei were uninvolved, except one small spot of reddish softening at the top of the lenticular nucleus. The internal capsule was firm and natural. From the ventricular surface the tumor extended to the head of the caudate nucleus, and the parts immediately behind it were softened as far back as the thalamus. Microscopic examination of the tumor showed the presence of tubercle. Attempts to demonstrate tubercle bacilli failed. As before stated, the lesions on the opposite side of the brain were hemorrhagic infiltrations, which occupied the lower divisions (crusta) of the left crus and the posterior end of the left optic thalamus.

Remarks.—The points of interest in the case which I have just detailed are manifestly the following: A large tumor, $3\frac{1}{2}$ inches long and $2\frac{1}{3}$ inches deep, occupied the white matter of the right frontal lobe as far back as the bases of the first and second frontal convolutions, and from the ventricular surface involved the head of the caudate nucleus, and by softening the structures as far back as the thalamus, and yet was practically, if not entirely quiescent. The convulsions, the hemiplegia, the conjugate deviation of the head and eyes, in fact all the cerebral manifestations that were present during the thirty hours which elapsed between the first convulsion and death, were evidently due to the fresh hemorrhagic lesions in the left crus and on the left optic thalamus. The optic neuritis, on the other hand, must have been present for some time before its discovery, indeed, it was probably the only symptom of the presence of this brain tumor.

The fact that optic neuritis was present in connection with this tumor of the frontal lobe is further interesting, inasmuch as it has been stated

that it is just in this region that tumors may exist without the production of changes in the end of the optic papilla. Dr. Gowers has well said, "Morbid appearances in the eye are frequent in intracranial disease, and often prove the existence of such disease when other symptoms are inconclusive, or afford evidence of its nature that can be obtained from no other source." The conjugate deviation of the head and eyes was in this case, as it is in a majority of instances, toward the lesion which produced the symptoms and away from the paralyzed side. The very reverse of this has been noted, and in Prevost's collection of cases, in three such exceptions, the lesion was found in the peduncles of the cerebrum or cerebellum. Here there was a hemorrhagic infiltration into the crus cerebri, but the usual order obtained. This tumor, faithful to its location in the so-called inert zone, produced no symptoms, and was, besides, too extensive in its invasion of the brain substance to have added any material or accurate data to the knowledge of its localization. For the very reason of its quiescence, however, it has seemed to be a useful and interesting case to report, especially in connection with the fresh and active lesions upon the opposite side of the brain.

I am indebted to Dr. John M. Taylor for the drawing of the base of the brain, and to Dr. Randall for the photograph of the child. Dr. William Osler kindly aided in making the autopsy and the subsequent dissection of the brain. *May* 26, 1887.

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15. *Embolism of left anterior cerebral artery; softening of left frontal lobe.*

Exhibited by Dr. W. Osler.

Thomas K. was admitted to hospital with fractured thigh and other injuries, the result of an accident. Death took place about ten days after admission with symptoms of coma.

Brain: The left frontal lobe tore on removal, and a soft puriform fluid exuded. This lobe looked flatter and was softer than the other. At the base vessels looked natural, a few thin plates of atheroma. On tracing anterior cerebrals, the left vessel, three-quarters of an inch beyond anterior communicating artery, was swollen, became tortuous, and was closely united to the inner face of first frontal convolution. At this point the vessel walls were thin, looked infiltrated, and of a grayish color. At the region of softening on the walls of the artery the two anterior cerebrals were adherent together. The vessel was plugged for

the length of three-quarters of an inch, firm except at the spot above referred to, where the walls were quite soft. The first frontal convolution, as far as its base, was softened. On section, there was a distinct puriform softening at the apex of the left lobe. In the white matter this extended back to the anterior horn, gradually becoming less. Superficially it involved the anterior part of the third, the gray matter of anterior portions of first and second convolutions not much involved. Small focus of softening in the inner section of left lenticular nucleus just above anterior perforated space. No further change noticed in brain.

There were no thrombi in the femoral veins. Heart normal. There was suppuration at the seat of the fracture. *June* 9, 1887.

VII. ORGANS OF THE SPECIAL SENSES.

1. *Mastoid disease; death from septicæmia.*

Exhibited by Dr. M. H. Fussel.

The specimens were removed from the body of Thomas M., æt. 25 years, stocking-weaver. Family history good. Never had scarlet fever nor any serious disease. Ever since vaccination at age of five years had had periodic attacks of suppurating middle ear with gradually increasing deafness. General health always excellent. The ear trouble was never treated except at times of greatest discharge. The patient was first seen by me on the evening of July 14, 1885. For several weeks previously he had been suffering from earache. Bowels had been constipated. Appetite and general health fair. On the previous evening (July 13th) had a slight chill followed by fever. On examination the hearing was $\frac{1}{48}$ in the right ear and watch only heard on contact with left. The right membrana tympani was entirely opaque, the left opaque in upper anterior quarter, the remainder of ground glass appearance and containing numerous small perforations. The left temporal region was swollen and tender, especially over the mastoid process, in this situation there was some redness. Morphia in the ear and poultices to the part were ordered.

July 15. Rested poorly all night, much pain ; temperature was 100°.

16*th*. Pain and tenderness better, but temperature 101°.

17*th*. A heavy chill last evening. Patient was evidently growing worse. Dr. R. R. Bunting, of Roxborough, was called in and a deep incision through the periosteum was made. Nothing but blood flowed from wound. After this incision patient was entirely without pain, but the general condition did not improve.

20*th*. In the evening a heavy chill, followed by a temperature of 104.5° ; the temperature dropped to 99.5° in a few hours, however.

21*st*. Temperature was again this A. M. at 102.5° ; patient becoming dull and heavy. Drs. Bunting and Hansel were called in. The old incision was extended. Just over the position of the end of the mastoid process pus was struck. It flowed in quite large amounts and was

exceedingly offensive. Dead bone was found at the bottom of the incision.

22*d*. A.M. temp. 101.5°. Patient bright and cheerful, no pain; pus flowing freely. P.M., another chill, pus had burrowed along course of sterno-mastoid for one and a half inches. The wound was probed and washed out and the patient relieved.

From this time, until death, on July 24th, the patient grew rapidly worse. He sank into a typhoid condition, from which he could not be aroused. Temperature remained about 103°. On the morning he died he vomited some bloody fluid and had a number of cutaneous hemorrhages. The blood would ooze through the skin, collect in drops, and, if not prevented, trickle over surface, veritable "bloody sweat." No head symptoms except some low muttering delirium.

Post-mortem examination, forty hours after death.—Body much emaciated. Foul pus issued from wound in mastoid region. The edges of wound decomposed. The skull cap having been removed, the meninges were found to be normal, except on the outer edge of the lobe of the cerebellum. The part that rested over the lateral sinus was slightly discolored; no actual inflammation, however. No abscesses in any portion of brain. A cut was made in the left temporal region of the skull, so as to embrace the mastoid cells in that region, and a wedge-shaped portion removed. On examining the anterior cut edge of the skull an irregular necrotic opening was noticed, it was about 3″ from the beginning of the osseous canal. A probe passed in the external meatus passed through this opening. In the portion of bone removed was an opening the counterpart of the above. It was an opening into the same necrosed portion of bone. The latter opening communicated with a large irregular cavity in the mastoid process filled with a pultaceous mass of necrosed bone. The entire mastoid process was diseased, all the cells being full of pus. The greater destruction of bone, however, was in the internal half, the external half of bone being comparatively healthy. Pus exuded from the numerous foramina in the digastric fossa; this was probably the source of the greater amount of pus during life. The lateral sinus did not contain a thrombus, but its walls were the seat of a purulent inflammation; the walls were greatly thickened and easily detached from the bone. The path through which the inflammation passed into the sinus was probably one of the mastoid veins. A vein of probably 1″ in diameter had its origin right in the midst of the necrosed bone and emptied into the lateral sinus. This vein was, unfortunately, cut through in the specimen.

The other organs of the body were normal, save in the apex of the

right lung were two small necrotic portions each the size of a bean, probably due to infarctions breaking down. The spleen and liver were free from infarcts and abscesses.

The septicæmia from which the patient died was not due to simple absorption of effete matter, but to actual admixture of pus with the blood. *November* 12, 1885.

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2. *Two cases of tumor of the orbit.*

Exhibited by Dr. G. G. Davis.

Robert D., æt. 37 years, colored, a porter by occupation. He gave the following history: Eight years previously, while chopping wood, a piece flew up and struck him on the edge of the orbit. His sight at this time was good and he soon forgot the accident. Four years later he got some lime in the same eye, and soon noticed that it protruded more than the right one. The protrusion of the left eye continued to increase, and nine months ago he lost all sight in it. He had no headache nor other cerebral symptoms and was otherwise in good health. On examining him the left eye was seen to be protruded; it was impossible for him to close the lid over it, and all motion and sight in it were gone. Surrounding the ball were several hard, lobulated tumors, particularly externally, both above and below. The skin was normal. Dr. Goodman, under whose care he was, removed the whole contents of the orbit down to the bone. It is now about two years since the operation and there has been no recurrence. Microscopic examination of the tumor shows it to be composed of dense fibrous tissue.

The other case is that of a child. Hannah M., æt. 3 years, came to dispensary with a tumor above the left eye. It was first noticed three months ago. In seven weeks' time it had reached the size of an English walnut. It was then removed by an incision through the mucous membrane. It recurred almost immediately and in six weeks' time reached the size of the first growth. On examination a round tumor was seen above the edge of the upper lid. It did not bind down the involved structures as the other tumor did, so that the lids could be nearly closed The movements of the ball were but slightly interfered with, exophthalmos was not marked, and the sight was good. The growth was very soft almost fluctuating, and the skin was translucent, showing the large and tortuous veins which ramified over the surface of the tumor. The

growth was removed by Dr. Strawbridge, and the wound healed, but recurrence soon took place and death occurred in about three months.

I have not brought these specimens before you on account of their rarity, because tumors of the orbit, benign and malignant, are common enough, but because we have here two typical cases and it may be instructive to compare them.

Concerning the origin of the growths I can say nothing positive. The orbit in the first case was completely filled, the growth even invading the muscles, and the muscular fibres can be distinctly seen in some places pervading the fibrous mass. From its mode of starting (a blow) it may have originated from the periosteum, certainly not from the eyeball itself. The tumor in the second case, from its situation, may have started from the lachrymal gland. It shows, under the microscope, as might be expected, a round-celled sarcoma. *February* 11, 1886.

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3. *A large sequestrum from the mastoid of a child.*

Exhibited by Dr. B. A. Randall.

The patient, Elizabeth C., æt. 2 years, was admitted to the Children's Hospital November 24, 1885, with suppurative otitis media on the left. There was a mastoid sinus and total left-sided paralysis of the face. Dead bone was found at the sinus and a sequestrum involving the mastoid process was soon after removed. The suppuration continued with little diminution until June, 1886, when the dead bone, which had been gradually becoming looser, was removed. This sequestrum consists of almost all of the mastoid portion of the bone not previously removed, including a portion of the outer surface measuring twenty millimetres horizontally, by fifteen vertically, and a part of its meningeal surface twenty-five by twenty millimetres. The entire mastoid antrum lies in the sequestrum, as well as the superior semicircular canal. The other semicircular canals and that of the facial nerve have been wholly destroyed. The most interesting point is that a square inch of the cerebral dura had been laid bare, as well as part of the lateral sinus, without serious result. The child is still doing well, though suppuration has not entirely ceased.

May 13, 1886.

4. *Microscopic specimens from a tumor of the conjunctiva.*

Exhibited by Dr. G. E. de Schweinitz.

Some few weeks ago I assisted Dr. J. William White in removing a small tumor from the conjunctiva of a man, æt. about 25 years. This small growth was situated on the right eyeball, between the retrotarsal fold and edge of the cornea, appeared like a mass of very vascular granulations, and was said to have originated in a burn of that spot by a spark from a cigar. The eye was highly hypermetropic and partly amblyopic. The sections show that externally the growth is composed of stratified epithelium, from this club-shaped papillæ of epithelium pass inward, and the centre is made up of delicate connective tissue and bloodvessels. There is, in other words, the structure of a papilloma, which has probably grown under the stimulus of a local injury. *February* 10, 1887.

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5. *Fibroma of upper eyelid.*

Exhibited by Dr. G. E. de Schweinitz.

A man, æt. about 35 years, consulted his family physician because of a small lump growing near the edge of the left upper eyelid. The growth appeared to be an ordinary chalazion. The physician incised it and cauterized it with a stick of nitrate of silver. Instead of subsiding in a few days, however, this spot took on an angry turn, became indurated, and its surface purplish in color. It resembled a pea-shaped keloid. Dr. H. R. Wharton then saw the patient, and excised the little mass with its skin covering. A section of the growth shows that the skin covering (mucous layer) is about normal, and within this are the elements of a slight connective tissue growth and numerous young cells, spindle and round. The growth evidently originated in the connective tissue above the tarsus, the components of which are not present. The young cells are probably connective tissue cells, and although the resemblance in places is closely that of a sarcoma, I think it is rather a fibroma. *June* 9, 1887.

VIII. TUMORS NOT OTHERWISE CLASSIFIED.

1. *A case of secondary epithelioma.*

Exhibited by Dr. G. Dock.

This case occurred in the practice of Dr. Keen, at St. Mary's Hospital, and I am indebted to him for this opportunity of recording it.

Mary C., æt. about 70 years, married, born in Ireland, applied for treatment November 29, 1884, when the following history was obtained: Personal and family histories seem to be unusually good. No traces of constitutional disease can be found. Uses alcoholic liquors in moderation and has smoked a clay pipe the greater part of her life.

Filling the right submaxillary region and extending up over the inferior maxilla was a tumor, the distinct outlines of which included a space about three inches in diameter. The lower part was very prominent, standing out as a flattened node one and a half inches in diameter and about three-quarters of an inch high, the whole height of the tumor being one and a half inch. The growth was hard, immovable on the lower jaw; the surface smooth, red, and changing to a dull purple on the nodular elevation. On the summit of this growth was an opening leading up and in for three-quarters of an inch. The skin around the opening was everted, and the surface of the crater-like cavity covered with large and small granulations, which exuded a thin, gray, offensive pus. The neighboring lymph glands were not enlarged. The general condition was good.

The patient stated that the growth first appeared six months before admission, as a "kernel" below the jaw. She applied various poultices and salves to it. The tumor grew rapidly until two months before admission, when it opened, discharging a large amount of pus. After that there was no apparent increase in size. She could assign no cause for the tumor, except a scald, received about one year before, on the lower lip near the angle of the mouth on the right side. This was followed by an ulcer which was removed at the Episcopal Hospital, in February, 1884, about two months before the appearance of the enlarged gland. Dr. J. M. Bradford, late Resident Physician at the Episcopal Hospital, informs me that the ulcer was noted as "epithelioma."

On December 3d, Dr. Keen removed the tumor, together with a margin of healthy skin and the submaxillary salivary gland. The external plate of the inferior maxillary appearing roughened, it was cut away. The cavity of the mouth was not opened. By the use of harelippins and sutures, the edges of the large wound (4½ inches in diameter) were approximated almost perfectly. The dressings at first were carbolized, afterward iodoform was used. In the fourth week after the operation a small, red, indurated, sometimes painful, spot appeared in the skin just posterior to the wound. A few days later the patient was discharged.

In the meantime sections were made through various parts of the growth, and showed the structure of squamous epithelioma everywhere. The salivary gland was invaded. No trace of the lymph gland could be found, and the supposition was that it had ulcerated away completely. The patient was lost sight of until the beginning of May, 1885, when she came to me with the following account:

After leaving the hospital the small swelling alluded to above, rapidly increased in size, and in a few weeks was larger than the one removed. She used no irritating measures, but the tumor broke down and ulcerated away, leaving a large granulating surface. On examination, I found an ulcer on the side of the neck, extending from one inch to the right of the median line to beyond the angle of the jaw, irregularly circular in outline, and containing islands of epithelium. There was a small opening into the cavity of the mouth, midway between angle and symphysis of jaw, and just inside of the inferior border of that bone. The symphysis of the jaw was drawn to the right about half an inch. There was a hard, tender swelling on the gum above the inner edge of the opening, covered with small, dark red nodules.

Early in June she was seen again. The inferior maxilla was still more atrophied, and had separated at the point of swelling and opening before mentioned. The adjacent end of bone and gum were covered with a small fungous growth. This process of atrophy and new growth is still continuing. The left alveolar process approaches the median line of the oral cavity, and the point of the chin is on a line dropped from the outer angle of the right eye. The ulcer on the neck is healing up, but the new growth in the mouth is rapidly enlarging, so that she cannot extend the tongue. There are no enlarged lymph glands, but within a few days she has complained of pain in a gland in the subclavian region. The general condition is very poor. The patient lives on liquid food, and takes morphia to produce sleep. *September* 10, 1885.

2. *Solid tumor of extensor tendon of the finger mistaken for thecal cyst.*

Exhibited by Dr. J. B. Roberts.

Dr. John B. Roberts presented a small tumor the size of a hickory-nut, which he had removed from the back of the wrist of a young man. It had the clinical appearances of the ordinary thecal cyst, or ganglion of the wrist. It felt like a tensely filled cyst of that character. Under this diagnosis it was punctured with a tenotome for the purpose of evacuating the contents and inducing cure. No fluid escaped; and then, suspecting its solid nature, Dr. Roberts transfixed it with a bistoury and split it open. A solid tumor, resembling in macroscopic appearance a fibroma, was then nucleated. It had evidently been developed within the theca of the tendon going to the middle finger; and probably between its fibres, for the movements of the finger were unimpaired, though a portion of the tendon was divided in removing the growth. On this account it was deemed unnecessary to perform tenosuture. The great variety of solid tumors in this location was mentioned, and the specimen referred to the Committee on Morbid Growths for microscopic examination.

Report of the Committee on Morbid Growths: "Your Committee reports that Dr. John B. Roberts's tumor removed from the theca of the extensor tendon of the middle finger, shows under the microscope the structure of a pure fibroma." *November* 12, 1885.

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3. *A case of retroperitoneal spindle-celled sarcoma with extensive thrombotic and hemorrhagic changes.*

Exhibited by Dr. W. Osler.

In addition to features of general clinical interest, the following case is worthy of record from the rarity with which spindle-celled sarcomata form large abdominal tumors, and still more so from the remarkable hemorrhagic destruction which the greater portion of the growth had undergone.

Michael D., æt. 60 years, machinist, resident of this country for thirty years, was admitted to the University Hospital, September 25, 1884,

with a tumor of abdomen. Had been a moderate drinker, had used tobacco to excess. Had been healthy, "never sick a day" until present illness. Family history good; had nine healthy children. About six months ago he noticed that the abdomen was getting large, but felt no inconvenience, and it was not until two months ago that he began to feel uncomfortable after eating, and began to lose flesh and strength. Had lost about eighteen pounds in weight. His appetite had, at times, been ravenous, and the thirst excessive. Bowels obstinately constipated, no movement without purgatives. No pain of any kind in abdomen, only an unpleasant fulness after eating.

Present condition: Moderate emaciation; weight 117 pounds. Complexion muddy. Temporal arteries prominent and tortuous. Tongue clean; appetite good, but he cannot take large meals on account of sensation of fulness. Says he is in good health, only weak. Passes about seven pints of pale urine of a specific gravity of about 1004, with a trace of albumen; no casts, no sugar. Examination of thoracic organs negative. Abdomen presents a symmetrical prominence in the neighborhood of the umbilicus, rather wide and flat. Superficial veins not distended. On palpation a solid tumor is felt, occupying the hypogastric and umbilical regions, and extending laterally into the flanks. It is irregularly nodular on the surface, slightly movable, and the rounded outlines above and laterally can be distinctly felt. Below, the outline is not clear and cannot be defined. Grasped firmly, it can be moved as a solid uniform mass, occupying a median position. No one part is softer than another, and there is no sense of fluctuation. Percussion gives a dull note over the tumor, tympanitic above in the epigastric region and in the lateral part of the umbilical. From spleen and liver the mass can be easily separated both by percussion and palpation. There is no pain in handling. Measurement round umbilicus thirty and three-quarters inches. Glands in groin not enlarged, feet not swollen.

He stayed in hospital until November 11th, the condition remaining unchanged, except that he gained three or four pounds in weight, and his general health had improved. The condition of the urine was of interest: for several weeks he continued to pass more than seven pints daily of pale urine of a low specific gravity, with a trace of albumen, but in the last three weeks in hospital the amount fell to about three and a half pints. On October 18th he was made the subject of a clinic, when the diagnosis of Lobstein's retroperitoneal sarcoma was made. The polyuria was attributed to irritation of the renal nerves caused by the pressure of the tumor.

On the 10th of November he went home. Subsequently he was

admitted to St. Mary's Hospital, under Dr. O'Hara, and the upper part of the mass, which had become soft, was aspirated by Dr. Mears, and a couple of quarts of bloody fluid removed. He was taken to his home, where he died in April, and I have to thank Dr. Miller, under whose care he was, for an invitation to be present at the autopsy, and for permission to utilize the specimens.

Autopsy, with Drs. Mears and Miller. Considerable emaciation; abdomen distended, discolored in upper part. A solid tumor could be felt occupying a large part of the cavity, firm below and soft above. On exposing the peritoneum a large mass occupied the lower three-fourths of the cavity, pushing up the intestines. The membrane was smooth, in places covered with small grayish-white nodules, and in the flanks there were a few adhesions. At the top of the tumor a coil of jejunum was adherent, and of a dark pigmented appearance. Many large veins coursed over the surface of the tumor, which was covered by the glistening peritoneal membrane. In the upper flaccid portion was an orifice, through which blood oozed, and in the peritoneal cavity there were about two pints of fluid of a similar character. The intestines were carefully removed; there was only the one adhesion, already mentioned. The mass, which was covered by peritoneum, occupied a median position, lying upon the spine, and extending to the brim of the pelvis. The hand could be placed behind it and passed down the vertebral column, and into the concavity of the sacrum, and in these regions the tumor had no adhesions. Below and to the left it was loosely united to the lower part of the sigmoid flexure, but the chief attachment was to the brim of the pelvis on either side of the fundus of the bladder, and particularly to the right side. The bladder itself was not disturbed in position; the top of it could be seen lying between the symphysis and the tumor. The adhesions, which could be readily separated, were closest to the transversalis fascia on the right side near the pubic bone. The weight was estimated at about eight pounds.

The tumor consisted of two parts, an upper cystic, flaccid portion, and a lower solid one. When opened, the cyst contained rather more than a pint and a half of bloody fluid, with flakes of yellowish-brown material, which also lined the wall, giving an appearance not unlike a large aneurismal sac. The walls were thin, and consisted of a peritoneal investment, within which was a firmer, condensed fibrous tissue. The blood removed at the tapping had evidently come from this sac. A transverse section across the solid part showed a central, firm, dry, yellowish-brown tissue, which cut with resistance, and which was everywhere surrounded by extravasated blood, occupying a position just within the capsule, and

in some places extending into the substance. In this section there was nothing suggestive of a neoplasm, the tissue resembled the dry, leathery contents of an old aneurism, except that there was no lamination. Section of the lower part of the mass near the pelvis revealed several soft, grayish-red portions, cerebriform or encephaloid in appearance, and evidently of a sarcomatous nature. They formed, however, a very insignificant part of the entire mass.

There was no special enlargement of the retroperitoneal or mesenteric lymph glands. The stomach and intestines presented nothing abnormal. The pancreas and spleen were unaffected. The liver contained one secondary mass in the left lobe, the size of an orange, and several smaller ones. The kidneys were fibroid, and the ureters and pelves dilated, particularly the right; due, doubtless, to pressure. The heart showed moderate hypertrophy of the left ventricle. Aorta smooth. Lungs much carbonized, and somewhat emphysematous. Brain not examined.

Histological examination: Teased portions showed that both primary and secondary growths were composed of large spindle cells, closely packed together. The remnants of the original growth situated at the lower part of the tumor were quite distinctive, and had not undergone degeneration. There were also, in some places, portions of sarcomatous tissue just within the capsule, separated from the central dry thrombus by freshly extravasated blood. Sections of hardened portions showed a typical spindle-celled growth. The thrombus presented a finely granular basis-substance, between strands of translucent, hyaline material. All traces of cell structure were gone.

Remarks.—The points of interest about this case may be briefly considered under the following heads.

The character of the growth: Spindle-celled sarcomata rarely form large abdominal tumors. In the examination of a considerable number of new growths of all sorts, removed from the peritoneal cavity, I have not met with a similar one.

The situation of origin was unusual. I fully anticipated that we should find it springing from the lumbar retroperitoneum, the common point of origin for large abdominal sarcomata. Here the growth seems to have begun in the subserous connective tissue in front of the symphysis, not from the peritoneum, for it was quite loosely attached. It is interesting to note that spindle-celled sarcoma not infrequently originates in the connective tissue of Scarpa's space, a tissue directly continuous with that from which the tumor in question grew.

The looseness of the attachment and the readiness with which the tumor could be lifted out of the abdominal cavity, made us regret that

we had not yielded to the patient's urgent solicitation to have the abdomen opened.

The character of the regressive changes: So soon as a tumor obtains any size we expect to find in it areas of degeneration, fatty, caseous, or calcareous; or, if a rapidly growing neoplasm, hemorrhages. Sarcomata are particularly prone to hemorrhage; indeed, when growing actively, it is rare not to find foci of extravasation in them. The effused blood not unfrequently becomes encysted, and the dark contents appear to result from the liquefaction of the coagulum. In a large tumor several such cysts may exist. Small scattered hemorrhages are more common, and the blood gradually undergoes changes without materially altering the appearance of the growth.

The condition in the case here reported is very unusual, as the greater part of the tumor had become converted into a dry, hard thrombus, while a considerable portion was occupied by a blood cyst, so that the mass resembled a huge hæmatoma rather than a neoplasm. Such general hemorrhagic destruction of a large tumor is not often met with, and I have not been able to find the record of a case with just such extensive thrombotic changes as here described. The mode of production can be readily understood from the appearances presented by the mass. No doubt softening and destruction by hemorrhage first occurred, as existed, indeed, at the upper part of the tumor, which formed a large blood cyst, the walls lined with thrombi, and in the fluid contents of which were flakes of firm fibrin. This cyst had been much larger, as nearly two quarts of bloody fluid were removed from it by aspiration. Had life been prolonged, the blood thus extravasated would, doubtless, have become inspissated by the absorption of the more fluid parts, and the remainder have been converted into just such a dense, dry mass as existed at the lower part of the tumor. It seems reasonable to infer that the firm, hard thrombus which constituted more than one-half of the entire tumor, was formed in this way. Possibly the hemorrhages beneath the capsule, which were very general, aided the process, though nowhere could a distinct lamination be detected.

The polyuria: Pressure of new growths in the abdomen upon the solar plexus or upon the renal nerves, may cause a very great increase in the flow of urine. The irritation in this case was transitory, as the amount fell to normal before he left the hospital. We do not yet know the precise conditions under which this occurs. Instances of it are rare, and we need careful observations on the state of the nerves. Dickinson, in his work on *Diabetes*, mentions a case in which degeneration of the solar plexus was found. Owing to the unfavorable circumstances under which

the post-mortem examination was performed, no dissection of the nerves could be made in this case.

Dr. J. E. Mears thought the growth could have been removed, though the removal would have been attended with some hemorrhage.

Dr. Tyson would like to ask Dr. Osler what, in his opinion, was the effect of thrombotic degeneration on the histological elements of tumors; and again, is it possible for clots to be converted into the tissue of the original tumor, as is asserted by some?

The President remarked that the case was of much interest from a clinical standpoint, in view of the possibility of surgical interference, and asked Dr. Osler whether the conditions, as found post-mortem, suggested any means by which such a tumor as this could be diagnosed from a similar growth occupying the more usual position in the lumbar region.

Dr. Tyson, in connection with the clinical history, called attention to a retroperitoneal sarcoma, presented by him to the Society last winter, which had been mistaken by him and others for a tumor of the kidney.

Dr. Osler, in reply to Dr. Tyson's first question, stated that the only remnants of sarcomatous tissue were two or three small, but very distinct, portions of the lower attached part of the tumor; the remainder had wholly undergone this thrombotic change, and in the upper part had become converted into a blood cyst. This change was, no doubt, slow, with first a destruction of the sarcomatous elements by the blood-clot, and then a slow process of necrosis. There was no evidence in any part of the tumor of an invasion of the coagulum by the sarcomatous elements, as is not infrequent in thrombi in other regions, as he had seen in the portal and renal veins. The chief interest in the specimen lies in the remarkable extent of the thrombotic change. Looking at the clinical aspect, he had diagnosed the case as one of retroperitoneal sarcoma from its large size, the central position, the slight movability, the distinct separation from liver, kidney, and spleen, not being placed more on one side than on the other, and from the fact that palpation in the lumbar region gave no pain or other evidence of kidney lesion. It was firmer above the brim of the pelvis than any other tumor he had ever examined. One remarkable feature about these tumors is their painless character; this patient complained of no pain, and in two other similar growths, which he described at length, pain was not a symptom.

January 14, 1886.

4. *Myxo-sarcoma of the scalp.*

Exhibited by Dr. C. B. Nancrede.

Mrs. S., æt. 25 years, native and resident of the eastern part of Kentucky, married, the mother of two children, the youngest 5 months old. Fifteen years ago first noticed a lump of "the size of a bean" on the back of the head. For thirteen years it did not enlarge and gave no trouble except when, after being bruised, it, for a short time, was swollen and painful. Two years ago it began to enlarge, and during the last pregnancy reached "the size of a goose egg." After delivery (five months ago) the mass grew rapidly and became very painful. When admitted to the Good Samaritan Hospital (Cincinnati), October 22, 1885, the tumor, pushing backward from the occipito-parietal region, was apparently as large as the head to which it was attached, measuring from before backward in the median line seventeen inches, in its median plane from side to side fifteen inches, in its greatest circumference twenty-one inches, and where it joined the head fifteen inches. There being no cranial or pericranial attachments, removal was readily and rapidly effected, and sufficient skin flaps secured to cover in the exposed surface. A part of the removed scalp is not at present attached to the specimen, having been cut off since the removal of the tumor for purposes of examination and study. The patient made a speedy recovery, and on the fifteenth day after the operation returned to her home, apparently perfectly well. The mass, three days after removal, weighed four and one-quarter pounds. *February* 25, 1886.

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5. *Specimens from a case of primary sarcoma in the anterior mediastinum.*

Exhibited by Dr. E. T. Bruen.

The specimen which I shall present to the Society this evening is a tumor taken from the body of Mary B., æt. 70 years. She was admitted to the Philadelphia Hospital June 26, 1886, and died June 29, 1886. At the post-mortem examination the body appeared well nourished. Lower limbs œdematous. The immediate cause of death was uræmia, consequent upon granular nephritis, but, in addition, the accompanying growth was found in the anterior mediastinal space. The tumor weighed

fourteen ounces; it was six inches long by five inches broad, and four inches in diameter. It was about the size and shape of the average normal heart. It presented a reddish-white, fleshy appearance, and was quite soft and pulpy in consistence. It was situated immediately behind the upper piece of the sternum, and consequently overlaid the aorta and pulmonary artery with the other structures lying in the deeper mediastinal spaces—it also encroached upon the left thoracic cavity. The upper lobe of the left lung was pushed aside by the mass in a direction outward and backward. It was splenified and the lower lobes of the lungs on both sides were somewhat congested. There were only a few slight adhesions between the tumor and the visceral layer of the pleura of the upper lobe of the left lung. The tumor was not in any manner attached to the sternal or costal bones. The bronchial glands in the anterior mediastinum were slightly enlarged, but the glands at the roots of the lungs were normal. The remaining structures in the thorax, including the heart, were normal. The kidneys presented the usual appearances characteristic of the granular contracted organ. The capsules were adherent and numerous cysts studded the cortical portions. The uterus was covered with fibroid growths, and the ovaries were calcified. The liver and spleen, with the other abdominal viscera, were carefully examined, but nothing of an abnormal character could be discovered. A biliary concretion was found imbedded in the intestinal mucous membrane just below the opening of the gall-ducts. The brain and its membranes were examined, but nothing of an unusual character was detected.

The specimen is, therefore, of special interest because it is an example of a case of primary sarcoma located in the anterior mediastinum without any secondary formation. As already stated, none of the lymphatics in the mediastinum were enlarged, and the growth seemed to have originated in the pleura of the anterior mediastinum. The results of microscopic examination would class this tumor with the small, round-celled sarcomata, with a tendency to alveolar formation.

The clinical features peculiar to this case, although by no means unique, yet deserve consideration.

1. The morbid growths in this region of the body are usually sarcomatous.

2. The age of the patient, which was advanced, is an unusual and yet recorded coincidence in tumors of a sarcomatous nature in this location.

3. The absence of wasting or cachexia. The patient's appearance was that of a person in average health. This peculiarity is also not remarkably exceptional in this class of cases.

4. The almost entire absence of any subjective symptoms pointing to the morbid growth. The patient was under observation but a few days before her death. She complained of slight cough and dyspnœa, and auscultation of the chest revealed feeble respiration over the posterior portions of both lungs, also anteriorly over the left apex. There was dulness on percussion over the sternum and some four finger-breadths to the left of the sternal margin. But when these signs were detected the patient was near her death, and their true significance was not established.

The patient came to the hospital for treatment of symptoms which were plainly dependent upon renal disease, such as albumen with casts in the urine, increased arterial tension, with the nervous symptoms usual in uræmia, which was the immediate cause of death. As the patient had worked as a domestic until shortly before applying to the hospital, it is fair to assume that there were no important subjective symptoms to call attention to the thoracic mischief.

5. The foregoing statement is in consonance with the history of anterior mediastinal growths, which are much less likely to occasion severe pressure symptoms than any other form of mediastinal tumor.

6. While the diagnosis in this case was not established during life, and while the case represents very forcibly the slight disturbance of the economy which may accompany certain of these mediastinal growths, and particularly those occupying the anterior mediastinum, yet it is quite probable that had there been opportunity for a more thorough and prolonged study the diagnosis might have been established. The dulness over the left sternal regions, the distant and feeble heart sounds, and weak respiratory murmur over the left apex, with the dyspnœa were certainly suggestive symptoms. *December* 9, 1886.

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6. *Sarcoma of the groin with secondary deposits in the lung.*

Exhibited by Dr. H. R. Wharton.

This tumor, probably sarcomatous in its nature, was removed from a patient in the University Hospital by Prof. Ashhurst. It was situated in the left groin and had existed about a year before he came under observation. The growth was attached to the sheath of the femoral vessels and surrounded the saphenous vein as it entered the femoral vein. In removing the tumor the saphenous vein was divided so close to the

point where it enters the femoral vein that to control the hemorrhage it was necessary to ligate that vessel above and below its junction with the saphenous vein.

The patient did well for ten days, experiencing no trouble from the ligation of the femoral vein, the collateral venous circulation being quickly reëstablished, but was then attacked with pleurisy, which caused his death upon the twelfth day after the operation.

The lungs removed from this patient show the presence of numerous secondary growths. The femoral artery and vein from the same case are also presented with the ligatures upon the vein *in situ*, the vein being filled with a firm thrombus. This specimen is of interest in connection with the treatment of wounds of the veins, in which accidents it has been recommended that the accompanying artery be ligated to diminish the amount of blood to be returned by the injured vein and thus prevent venous stasis with its accompanying dangers. *March* 24, 1887.

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7. *Epithelioma of hand.*

Exhibited by Dr. H. R. Wharton.

This large epithelioma of the hand was removed by amputation at the lower third of the forearm by Prof. Ashhurst, from a woman, æt. 60 years, who was a patient in the University Hospital. The primary growth had been removed about a year previously. The wound healed promptly, but the growth recurred some time after she left the hospital. When she presented herself again at the hospital the growth had attained the present dimensions and the patient was being worn out by the pain and profuse hemorrhages, so that it was deemed advisable to remove the growth by amputation of the forearm. *March* 24, 1887.

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8. *Osteosarcoma of the forearm.*

Exhibited by Dr. J. B. Deaver.

The growth, of the size of a hen's egg, involved the lower extremity of the left ulna and was removed by resection, the bone being sawn through at some distance above the growth. The use of the arm, six months after the operation, was perfect. There had been no recurrence. The patient was exhibited to the Society. *May* 12, 1887.

9. *Cystic goitre originating in an accessory thyroid gland.*

Exhibited by Dr. J. B. Deaver.

The patient from whom this growth was removed was a woman, æt. 23 years, who for nine years had noticed a swelling in her neck. It gave her no pain unless she swallowed a very large bolus of food, was deep seated, and moved up and down with swallowing. At the operation the tumor was found covered with a plexus of tortuous veins and was adherent to the trachea, œsophagus, vessels, and spine. It was impossible to disconnect it from the right lobe of the thyroid gland, which was removed with it. The right lobe was larger than the left and near the tumor was deeply pigmented, having a decidedly different appearance here from that of the remainder of the lobe, which was normal. On section the tumor was found to consist of thyroid tissue in a state of cystic degeneration and had probably been originally an accessory thyroid gland. *June* 23, 1887.

IX. SPECIMENS FROM THE LOWER ANIMALS.

1. *Tænia echinococcus.*

Exhibited by Dr. W. Osler.

This rare parasite was reared experimentally by feeding a dog with hydatids from the liver of a pig. The animal was killed about seven weeks after the feeding when the intestine was found to contain many hundreds of the mature tapeworms. The portion of bowel exhibited had many adherent to the mucous membrane. From the small size of the worms, only a few lines in length, they are apt to be overlooked. Cobbold states that the only specimens procured in England have been experimentally reared. Dr. Leidy has never met the adult worm in this country. That it may occur here in the dog is very evident from the frequency with which echinococcus cysts (the larvæ) are met with in the hog and other animals *September* 24, 1885.

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2. *Cysticercus cellulosæ of brain, heart, and voluntary muscles.*

Exhibited by Dr. W. Osler.

The "measle" or cysticercus of the Tænia solium is found very frequently in the hogs at the Berlin abattoir, and these specimens illustrate the extraordinary numbers which may exist in a well nourished and apparently healthy animal. The brain presents at least thirty or forty, all encysted, and very readily seen as opaque white bodies the size of large peas. It is remarkable that the animal does not, even with such numbers, have symptoms of brain disease. I suppose the slow growth of the parasites and the comparatively small size to which each one attains permits of the displacement of tissue without much disturbance of function.

In the heart the cysts are also very numerous and at least a dozen may be counted beneath the endocardium of the left ventricle. They

were very generally distributed throughout the voluntary muscles and in this case the diagnosis of the condition could be made with half a glance. When scanty even the most careful examination may fail to detect them, and probably the chief danger is in these cases, as, of course, a single cysticercus taken in a slice of raw "schinken" or in "wurst" is sufficient.

In this case parasites were so numerous that I made a careful inspection of the mouth to see if their presence could not be determined in the lingual and buccal muscles. Beneath the tongue three or four could be distinctly seen, and the affection might really have been diagnosed during life in this way. As with the trichina, the hog appears to harbor the cysticerci with very little inconvenience and the symptoms of the affection are very indefinite. In this country pork "measle" is not nearly so frequent as the veal and beef form—the larva of the T. mediocanellata. *September* 24, 1885.

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3. *Liver, with dilatation and calcification of the bile-ducts, the effect of flukes.*

Exhibited by Dr. W. Osler.

The liver fluke, Distoma hepaticum, so common in Europe, is not very often met with in sheep and cattle in this country, and in my experience it is rare to find here the advanced changes described in works on parasites. When in Berlin, in 1884, I spent two afternoons of each week at the abattoir, which, owing to the elaborate system of inspection, both ante- and post-mortem, offers one of the best fields in Europe for the study of comparative pathology and helminthology, and through the kindness of Dr. Hertwig I was enabled to secure a large number of interesting specimens.

The liver here shown represents a condition which may be seen almost any day. The organ was not much enlarged and externally did not look much altered, though here and there a gray-white spot could be seen. I have dissected the bile-ducts of the right lobe from the under surface, leaving intact the upper portion of the liver substance. The appearance is not a little remarkable. The ducts are uniformly enlarged and converted into rigid, calcified tubes. The primary branches are as thick as the thumb and the terminal ones toward the edge of the liver the size of pipe-stems. When broken the wall is from one to two lines in thickness, calcified, quite brittle, and without a trace of any tissue.

Here and there are saccular dilatations. The inner surface is roughened and irregular, stained of a black or yellow-brown color, and in the larger tubes there were a few flukes. In other specimens I have found them in numbers, dead, broken up, and calcified. A bile-stained glairy mucus filled the tubes. The condition is one of chronic cholangitis due to the irritation of the flukes. It is interesting to note that the liver substance is not much changed, and is not to any extent cirrhotic. The animal from which this specimen came was fairly well nourished, sufficiently so to escape the vigilant inspectors. In sheep the changes in the liver may be much more advanced and the symptoms of the affection, known popularly as the "rot," closely resemble those of cirrhosis of the liver in man. *September* 24, 1885.

X. MISCELLANEOUS.

1. *Fœtus papyraceous; fœtus compressus.*

Exhibited by Dr. J. H. Musser.

At a late meeting of the Chicago Medical Society, Prof. Etherington exhibited a specimen of the above character, and observed its rarity—but five cases being referred to in the *Index Medicus*. Prof. Jaggard referred to the literature of the subject, and said that most German obstetricians referred to this form of fœtus in their works.

I am indebted to Dr. Whiteside, in whose practice the case occurred, for the exhibition of this fœtus. The patient was delivered in the morning of a mature fœtus. In the evening he was called to relieve the patient on account of severe pain, attributed by the nurse to a full bladder. On examination he found the arm of this fœtus presenting, and with difficulty he succeeded in delivering. The patient did well.

You will observe that we have here a fœtus of the fifth or sixth month of pregnancy. With the exception of shrivelling or pealing of the skin, it is quite normal. The cord is thin and soft; its length not known. The placenta is thin, flat, and compact, it is subdivided as usual, but each part is whitish-yellow, presenting a uniform fatty degeneration. I desire to report further on the microscopical characters. The causes of the fœtus compressus are detailed in the paper referred to and published in the *Boston Med. Journal* of the present year. The interesting question of superfœtation is also discussed in that article.

November 2, 1885.

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2. *The "pig-backed" or alcoholic kidney of drunkards; a contribution to the post-mortem diagnosis of alcoholism.*

Read by Dr. H. F. Formad.

I wish to invite attention to a certain peculiarity of the kidneys of drunkards. It concerns the gross anatomy and appearance of these

organs, and chiefly their shape. A certain constant naked-eye appearance of the kidneys of hard drinkers on the autopsy table appears to me diagnostic of alcoholism. It is due to a chronic venous, or passive congestion, leading to a cyanosis and sometimes to œdema of the kidneys, giving to these organs a peculiar swollen, rounded shape, which I am in the habit of designating as the "pig-backed" kidney. It differs essentially from the renal cyanosis of cardiac disease. It differs also from Bright's disease, and I believe it to represent an independent kidney affection.

While I have been pointing out this "pig-backed" kidney for some time in my laboratory demonstrations, and lately have repeatedly shown specimens of it in connection with other lesions of alcoholism before the Society, I have not as yet published a detailed account of this peculiar and fatal kidney affection. I am not aware that any of the authorities on renal diseases have studied or described this kidney lesion of drunkards, which I believe to be a quite interesting and significant one.

A word of comment upon the character of the material that has been under observation is necessary. I do not intend here to consider the general morbid anatomy of the kidneys of the universal "moderate" drinkers, since these individuals eventually die of any kind of ailments and with any kind of kidneys, often dying from exposure, carelessness, and irregularities in diet and life incident to the state of drunkenness, and perhaps not at all from the direct effects of alcohol.

The class of cases which I shall chiefly consider in this communication is one to which the coroner is more often called than the practising physician. I have selected only those cases where the person died more or less suddenly, where the history and general appearance clearly showed that death was due directly to alcoholism, and where all other factors in causing death had been carefully excluded as far as possible. I have particularly excluded such cases in which the persons had primary heart disease and other affections known to produce renal cyanosis. Most of the subjects had been in good general physical health, but having been inveterate drunkards and nearly always intoxicated, died while on a debauch, or, in rare instances, after a brief illness following it.

Through the kindness of the Coroner of Philadelphia, who, of necessity, is called upon to issue death certificates in such cases, I have had, during the last two years, the opportunity of making observations which some time ago numbered *two hundred and fifty cases*, belonging strictly to the category above referred to, and mainly from my own autopsies.

In this city with a population of about one million, there are annually nearly two thousand cases of sudden death subject to legal inquiry. The proportion of cases of alcoholism appears thus remarkably large. I am indebted to my colleague, Coroner's Physician Stewart, for some of his autopsy material and his corroboration of the "pig-backed" kidney, and to Drs. George H. Chambers and A. Smith for valuable assistance in these observations.

A. Anatomical considerations.—I have met with two varieties of the kidney lesion now to be described:

First, a hard cyanotic form of the "pig-backed" or alcoholic kidney, in cases of sudden death; and

Second, a soft œdematous form of the same, in cases where death had been delayed.

First form: In nearly all persons who died more or less suddenly from the direct effects of alcohol, I found the kidneys to present the following appearances: The kidneys are always above the normal size; they are often from one-third to one-half larger, and are sometimes even double their usual size and weight. They are longer and thicker than normal, while their width is diminished, so that the natural, characteristic flat kidney, or bean-shaped form, is changed to a swollen, rounded, sausage-like or "pig-backed" appearance or form. They are bluish-red or livid in color from being engorged to their utmost capacity with venous blood, reminding one of the rounded enlarged appearance of animal kidneys when overfilled by some artificial injection mass. In most cases these "pig-backed" kidneys are elastic, but quite hard when freshly removed from the body; but unlike the cyanotic induration from heart disease, they gradually become softer, unless the renal vessels are at once tied to prevent the escape of blood from them.

On section the cut surface presents the same dark red or livid appearance as seen in the cardiac form of renal cyanotic induration. While in the latter, however, the pyramids of the medullary substance are especially congested, in the alcoholic cyanosis the whole kidney substance is almost uniformly dark red. Dark blood oozes from the cut surface and blood extravasations can be often seen by the naked eye, principally below the capsules.

Second form: The other form of the alcoholic kidney referred to, was met with in persons who had died some time after a debauch (suffering from a few hours to a few days from delirium tremens previous to death). The kidneys of such subjects are found to be soft and flabby from œdema, and they are less red; otherwise presenting the same appearance as the

hard variety of alcoholic renal cyanosis, including the "pig-back" shape of the kidneys.

In either form of this kidney lesion the capsule strips off easily, the surface is smooth, but often lobulated, not unlike the lobulation seen in congenital conditions. If the alcoholic lesion is not complicated by heart disease, the "pig-backed" kidney is less heavy in proportion to size, and less firm and tough and also less dry than the kidneys of the pure cardiac renal cyanosis. This is probably due to the greater amount of blood present and to the accompanying œdema, which I have not seen in the cardiac variety.

In the purely cardiac affection, however enlarged, the kidneys retain their natural flat shape. This may be explained by the fact that in the latter case morbid change is slow, gradual, and uninterrupted, and the resulting change (the induration from the connective tissue overgrowth) is a permanent one—whereas in the alcoholic cyanosis the process is rapid and intermittent, the effects of the alcohol being paroxysmal with each debauch and the mere resulting congestion receding more or less during the intervals of sobriety. It is probable that in the latter case the kidneys may return temporarily to their normal condition and shape.

A transverse section through the middle of the kidney from a case of alcoholic cyanosis is sometimes nearly circular, while a similar transverse section through a normal kidney or one of a cardiac renal cyanosis is oval or wedge-shaped and at least twice as long as broad.

Both kidneys are equally affected, sometimes the upper ends are found to be flattened or compressed anteriorly from pressure exerted by the enlarged liver on the right, and an enlarged spleen on the left side; the left kidney, as a rule, was more often found to be of a typical "pig-backed" shape than the right, and more frequently compressed at its upper end.

One hundred specimens of these kidneys were weighed and revealed the average weight of 250 grammes, or a little more than seven ounces —about two ounces over the normal average weight. The average variation in size and weight between the right and left kidney was insignificant.

A certain proportion of the 250 cases included in my records showed, beside the alcoholic renal lesion, appearances of Bright's disease. It is difficult to draw in all instances a definite line between these two lesions, but I would say that in about 10 per cent. (or in 25) of the cases Bright's disease was evident. In 16 of these cases chronic catarrhal nephritis with formation of small cysts was noted; and in 9 cases chronic inter-

stitial nephritis or a tendency to the granular contracted kidney was apparent. But, however affected otherwise, the "pig-backed" shape of the kidneys was always a conspicuous feature in these cases. It appears that this chronic venous congestion (and œdema, when present) of alcoholism affects structurally diseased and normal kidneys in the same manner and manifests itself alike in both.

B. The cause of the "pig-backed" shape in the alcoholic renal cyanosis.—The reason why the alcoholic kidney presents the roundish swollen shape is because the organs are over-distended with blood and serum, the kidneys acquiring the properties of, and in extreme cases may be compared with, true erectile or cavernous organs.

A "pig-backed" shape of kidneys can easily be induced experimentally in animals when the renal veins are tied or when kidneys removed from the body are artificially injected with a liquid.

Experiments have also shown that a hypertrophy of one kidney follows the removal of the other kidney, and under these circumstances I have seen the remaining kidney enlarge to double its natural size and acquire a typical "pig-backed" shape from being overfilled with blood.

A nearly similar appearance of hyperæmia of the kidneys may be seen in cases of acute fever, particularly in cases of infective fevers of children when death occurs during the height of the attack.

The loss of the flat kidney shape may be compared also with the loss of the biconcavity of red blood disks or corpuscles, which when immersed in any liquid which they absorb become spherical bodies.

C. Microscopy.—Sections were made from a number of specimens of the alcoholic kidneys, care being taken to examine microscopically all the different structural parts of these organs. For brevity's sake, only the essential changes will be referred to.

The cortical and medullary portions of the kidney appear to be similarly affected, but, as a rule, the latter less so than the former.

In the hard, cyanotic form of the alcoholic renal cyanosis the Malpighian glomeruli of the cortical portion are highly congested, and extravasated blood is seen within many of the Malpighian capsules; some are enlarged to double their normal size, but the majority appear rather compressed by the surrounding swollen uriniferous tubules and by their over-distended bloodvessels. The latter (both arteries and veins) show thickened walls and are deeply congested nearly everywhere throughout the organ. The stellate veins in particular are enormously distended and plugged up by blood-corpuscles as if thrombosed. The lymph spaces beneath the capsules as well as around the tubules are also dilated and distended, and are either seen empty or they contain extravasated

blood. In some sections the kidney structure gives the appearance of a cavernous change.

The epithelium of the convoluted tubules is cloudy, swollen, opaque, and the nuclei obscured by granules, which, however, become cleared up upon the addition of a solution of sodium hydrate. The epithelial cells appear to be double their normal size and although no visible desquamation or proliferation of the cells could be noted in any one of the typical specimens examined, it appears that the lumen of the tubules is obliterated completely, as seen in transverse sections of the tubules.

In the straight tubules of the medullary portion the same changes have been noted, but are less marked.

The connective tissue elements of the pure alcoholic kidney show no pronounced hyperplasia. In the sections of most of the kidneys it is hardly perceptible. Sections in which the epithelial lining was forcibly removed by means of a camel's-hair brush showed the connective tissue to be dense, stiff, often pigmented, but only moderately increased in a few places around the bloodvessels.

The connective tissue increase was notably prominent only in sections derived from occasional specimens which showed at the same time other evidences of inflammatory changes (Bright's disease), and in cases complicated with heart disease. I do not remember having seen in one of the sections of the pure alcoholic affection tube-casts or blood within the uriniferous tubules.

In sections from the soft, œdematous form of the alcoholic kidney, the minute changes were essentially the same, but not so marked as those described above. Extravasations of blood were less frequently seen, while the connective tissue elements appeared occasionally more or less proliferated. In sections from some cases, however, no other change could be observed than the very prominent cloudy swelling of the epithelium peculiar to all the specimens of the alcoholic kidney.

I do not think it necessary to describe the sections from the few cases in which Bright's disease and the alcoholic renal cyanosis coexisted.

D. Pathology.—From the above studies it appears that the exact position of the "pig-backed" or alcoholic kidney in morbid anatomy is as follows: There are, as stated before, two forms or perhaps stages of this lesion. *First*, a *chronic or venous congestion with cloudy swelling*, or a renal cyanosis with hypertrophy represented by the *hard, red, "pig-backed" kidney* (the earlier and more active lesion); and *second*, a *hypertrophy with œdema* of the kidneys, represented by the *soft "pig-backed" kidney* (the more latent and less rapidly fatal lesion). The latter I met less frequently than the former, and believe it to be the later stage of

the alcoholic kidney. Whether the lesion described eventually develops into Bright's disease if the victim survives, I am unable to tell from my purely post-mortem observations.

I met with only twenty-five cases of established Bright's disease among two hundred and fifty cases of confirmed drunkards, from which it appears that Bright's disease is not more frequent in drunkards than in any other class of patients. Some of the foremost clinicians and authorities on kidney diseases (Bartels, Rosenstein, Tyson, etc.) are disinclined to give a prominent place to alcohol as a causative factor of Bright's disease, which appears to be in accord with the above observations.

That either form of the alcoholic renal cyanosis may be fatal in itself, without terminating in Bright's disease, I have satisfied myself, and hence I *believe that it deserves a place as a separate and independent kidney affection.*

The true mechanism of the *death* of drunkards from this alcoholic renal cyanosis is a matter of conjecture. If causes such as heart failure and œdema of the brain (which probably were the ultimate, immediate causes of death in some of my cases) are eliminated, a fatal uræmia is then the only plausible explanation. The empty urinary bladder in some cases, the high specific gravity of the urine, when found at all, post-mortem, in the bladder, and the histories which I had obtained in a few of the cases, favor such a view. In many cases of sudden death of drunkards it appears that death must have ensued under conditions similar to asphyxiation, because the blood in these cases was found dark and liquid throughout the system. A like condition of the blood, however, I have seen in certain other forms of poisoning, such as chloral, hydrocyanic acid, and carbolic acid. This subject requires further study.

But, while I have seen hundreds of drunkards dead of alcoholism, thousands of equally hard drinkers are at large and well. It appears that under favorable conditions the alcoholic renal cyanosis is capable of returning to the normal state of the kidneys, and that in its early stages it may be only a temporary lesion, paroxysmal, like the excessive drinking. Quite significant seems to me the experience of the drunkards themselves. One of them, a brewer, who had been a number of times prostrated from alcoholic excesses, and whom I treated about ten years ago for delirium tremens, told me that of late years he keeps himself well, because he stops drinking beer at once for a day as soon as he fails to urinate about every hour or two. I examined his urine lately and found no traces of renal disease.

E. Pathogenesis.—The ordinary cyanotic induration of the kidneys, the result of long-continued permanent passive congestion of these organs, caused principally and directly by organic heart disease and indirectly by aortic and pulmonary affections, has been recognized as a separate kidney affection since the time of Traube. Tumors pressing upon the vena cava or upon the renal veins, or thrombosis of the latter, also produce renal cyanosis. So I should think any causes interfering with the return of blood from the kidneys will produce renal cyanosis, even if the cause resides in the kidneys themselves. The kidneys are organs especially predisposed to congestion, on account of their close and direct vascular communications with the large abdominal blood channels, and the renal veins, as Tyson well puts it, "are without valves and are the first, therefore, to receive the brunt of stagnation."

The alcoholic or "pig-backed" kidney we have seen to be essentially a renal cyanosis also, only differing, as pointed out above, in some anatomical features from the known cardiac renal cyanosis. The essential etiological factor, however, is in both the same, namely, the chronic venous congestion. The question is only as to mechanism of the production of the alcoholic cyanosis. In alcoholism we must look to the kidney substance itself for the cause of the obstruction to the renal circulation, because there is, as a rule, no cause for it outside of the kidney; no disease of the thoracic viscera and no thrombosis of the veins. Of the latter fact I have satisfied myself from the examination, in a number of cases, of the vena cava and the renal veins. Besides, in most of my rapidly fatal cases of alcoholic cyanosis I found the blood to be non-coagulable as stated before. It is possible that the large, fatty liver, which sometimes reaches an enormous size, may be held accountable for the pressure upon the large abdominal venous trunks; but I have often seen "pig-backed" kidneys in drunkards with but moderate hepatic enlargement.

I have heard Virchow say that "beer drinkers have *hypertrophied* kidneys." This view is also expressed by other pathologists, but no explanation is offered, nor have the anatomical peculiarities of the kidneys of drunkards been described. In relation to the pathogenesis of the alcoholic renal cyanosis the following explanation suggests itself:

It is well known that in persons who ingest great quantities of fluid, particularly alcoholic beverages in dilute form, the kidneys are strained to overwork. The quantity of urine normally passed by such persons is enormous so long as the kidneys act at all, and can at times be favorably compared with the quantity of urine passed in diabetes. It is evident that such overwork must invite a constant active (arterial) hyper-

æmia which of necessity produces a hypertrophy, due to a cloudy swelling of the epithelium and an overgrowth of all the structural constituents of the kidneys. A long continuance or a constant repetition of these conditions leads eventually to a passive (venous) congestion, which persists or increases or subsides according to the repetition and the duration of the debauches of the drunkard. The renal circulation of the blood is retarded by the pressure of the swollen epithelium of the uriniferous tubules as exerted upon the vessels—a pressure which the thicker walls of the intertubular arteries and arterioles can resist more readily than the soft and thin walls of the corresponding veins. There appears to be also thrombosis of the stellate veins. The exit of blood is thus retarded while the arterial pressure continues unabated and keeps the kidneys constantly overfilled with blood. Eventually the kidneys are filled to their utmost capacity with blood overcharged with carbonic acid, and in consequence also with serum, which, leaking out of the obstructed veins into the renal lymph spaces, makes the kidney tissue œdematous; blood extravasates also, and adds to the firmness, redness, and roundness of the organ. Finally, the renal circulation comes to an entire standstill, coincident probably with a suppression of the urinary secretion.

This seems quite evident from the post-mortem appearances and from the swollen, cyanotic, and sometimes œdematous "pig-backed" kidney of drunkards, and is suggested also from the observations derived from the microscopical examination of sections of these kidneys.

I will return to these studies in a future paper.

F. Statistical remarks.—Of the 250 cases of sudden death from alcoholism so far analyzed, 176 were men, and 74 women. The hard, red form of the "pig-backed" kidney was found in men, principally in the younger and middle aged; while the softer œdematous form was more common in men of advanced age and in the women. The kidneys in which Bright's disease was coincident with the alcoholic lesion were mostly those of persons in advanced life.

The alcoholic renal lesion was nearly equally divided between persons of Anglo-Saxon and German descent, and, so far as I could perceive, equally between whiskey and beer and ale drinkers. I cannot speak about wine drinkers who in this country belong to the higher classes of society, and are seldom reached by the scalpel of the pathologist.

It is interesting to note in connection with this point that the large, fatty liver (occasionally more or less indurated, but not contracted) which I met with indiscriminately in nine-tenths of all cases of death

from alcoholism was equally distributed among all the denominations stated above, imbibers of strong and of dilute alcoholic beverages.

The following order in the occurrence of the important and more frequent lesions may be roughly given from my autopsy records of 250 persons who died from alcoholism strictly:

The "pig-backed" or alcoholic kidneys	248 times.
Fatty, infiltrated, enlarged liver	220 "
Cystitis, acute and chronic	170 "
Mammillated stomach (or chronic thickening of walls)	150 "
Congestion and œdema of brain (meningeal and ventricular)	150 "
Simple cardiac hypertrophy	90 "
Atheroma of vessels and valvular heart disease	50 "
Acute gastritis	50 "
Bright's disease (various forms)	25 "
Phthisis	20 "
Cerebral apoplexy	10 "
Cirrhosis of liver, with contraction below normal	6 "

I will make no comments upon the above figures in the present communication. The lesions of alcoholism other than the "*pig-backed*" kidney, I will consider on another occasion. Of great interest are the changes in the stomach, liver, and brain.

The brains of confirmed drunkards should be studied by psychologists. The claim that chronic drunkenness is a form of insanity may perhaps be found to have some foundation.

G. The medico-legal aspect.—A thorough knowledge and correct interpretation of the post-mortem appearances of alcoholism is perhaps to no end more important than when the life or welfare of a fellow-creature is at stake. There is a great deal to learn in this direction generally, and in no department of medical science are text-books more indefinite and "behind the times," and more rich in errors, than in the subject of medical jurisprudence.

I have myself once testified in court in an insurance case—and I know of other physicians who have done so—that "a certain person who had a large, fatty liver, to our best knowledge and belief was not likely to have been a hard drinker during life, chiefly because at the autopsy no cirrhosis of liver was found!" This was fully in accord with the teachings of our masters in medicine and with text-books. Yet, I am sorry to say, it is entirely wrong; and the jury decided against the insurance company on account of such erroneous medical testimony.

My experience has since taught me that cirrhosis with contraction of the liver is at least as rare an affection in drunkards as it is in "teetotalers," and that the traditional "hobnail" or "gin-drinker's" liver is not diagnostic at all, while the large, fatty liver is one of the most prominent signs of alcoholism. The facts are that in 250 drunkards I found the enlarged, fatty liver 220 times, and the contracted, cirrhotic liver but 6 times.

Often when a drunkard falls dead on any occasion, a cry of murder is raised, especially when bruises are found upon the body. It is here that a familarity with the post-mortem appearances of *fatal* alcoholism is particularly necessary and nowhere can ignorance on the part of the examiner do more harm than here.

The constant occurrence of "*pig-backed*" or *alcoholic* kidney in hard drinkers who perished from their drinking, and the rarity of kidneys of such character in those who are not confirmed drunkards, induced me to regard the kidneys as a valuable sign in post-mortem diagnosis; and next to the presence of alcohol in the stomach, they are, together with the large, fatty liver, the most valuable proof that alcohol was operative or had contributed in producing death.

The "pig-backed" or alcoholic kidney, while a valuable diagnostic sign of the *effects* of the prolonged abuse of alcohol, may not show itself in persons who had been moderately addicted to the use of alcohol, and in such cases alcoholism should not be given as *the* cause of death.

Dr. Tyson could not speak from a systematic observation of a large number of autopsies in the cases of confirmed drinkers, but he remembered distinctly being surprised in several cases by the absence of cirrhosis where he confidently expected to find it.

Dr. Wilson said that Anstie, in the article on "Alcoholism" in Reynolds's *System of Medicine*, had called attention to the comparative infrequency of contracted liver in confirmed drinkers. This observer in an extensive out-patient practice in London, had seen large numbers of cases of alcoholism, but very few among them presented the physical signs of cirrhotic (contracted) liver. The experience of the staff at Blockley Hospital sustains this view. There many of the patients are soaked with alcohol, but even among those whose death is directly or indirectly due to alcoholic excess, fatty liver is much more common than contracted liver.

Dr. Osler thought the experience of pathologists and morbid anatomists with histories of patients is not of the most satisfactory character, he often having had cases to dissect where he knew very little of the history. Before saying these cases were chronic alcoholics, Dr.

Formad should present more specific statements about them. His own experience with livers in a large number of autopsies on cases of chronic alcoholism had led him to divide them into four classes: 1. Those in which the condition of the liver is pretty satisfactory; some of these cases may take alcohol for many years and yet the liver pass muster. 2. Fatty cirrhotic livers; the cirrhosis may not, perhaps, be distinct to the naked eye, but plainly shown by the microscope; this is the largest class. 3. Hobnail livers; these he would say were much more common than in Dr. Formad's series. 4. Hypertrophic cirrhotic livers. The difference between his observations and those of Dr. Formad might possibly be accounted for by a difference in the form of alcoholic beverage taken. He had not observed the special form of kidney described by Dr. Formad. In reply to a question, he said, in order of frequency he would place the livers, fatty cirrhotic, hobnail, hypertrophic cirrhotic, apparently normal.

Dr. S. Solis-Cohen said there were several theoretical considerations which suggested themselves in this connection. The text-books teach that the lesions of alcohol are of two kinds, sclerosis and steatosis. It is known that in some organs the fibrous change precedes the fatty one. The latter is the higher grade of degeneration. The subjects of Dr. Formad's autopsies were confirmed whiskey soakers, in whom one would expect to find more intensity of degeneration than in those whose use of alcohol, though persistent and excessive, was not so outrageous. Another point which had not been attended to, was the fact that some lesions might result from a local action of the poison upon the tissues—while others might be due to its systemic action. No study of the subject could be complete in which these points were overlooked.

Dr. Randall suggested that the point touched upon by Dr. Osler—the character of alcoholic beverage—might be very important. In Vienna, among beer drinkers, he had found the fatty liver much more common than the cirrhotic; while in England, where much gin is drunk, and he should suppose in Scandinavian countries, where they drink altogether strong spirits, the cirrhotic liver is doubtless comparatively frequent.

Dr. Musser had recently had to go over the records of the Pathological Society, especially in liver diseases, and had found the total experience of different observers the same as Dr. Formad's, and also in those cases cirrhosis was caused not so much by heavy drinking as by persistent drinking of spirits on an empty stomach.

November 12, 1885.

3. *Remarks on the bacillus of lepra; microscopical preparation.*

Exhibited by Dr. J. P. C. Griffith.

The specimens, from which the preparations shown this evening have been made, are chiefly from a case which occurred in Demarara, but of whose clinical history I can learn nothing. There is also a slide of material which I obtained in Vienna.

Some understanding of the pathological histology of the disease is necessary before studying the nature and distribution of micro-organisms.

The infiltration of lepra consists of granulation tissue much resembling under the microscope that of lupus, except that it is not so much divided into nests as in the latter affection, nor is there so great a supply of bloodvessels. The deposition of lymphoid cells takes place primarily in the corium, and extends thence along the course of the bloodvessels toward the surface and into the deeper layers of the subcutaneous tissue. The production of giant-cells and the occurrence of retrograde metamorphoses are to be observed both in lepra and lupus. A preparation from one of the nodules of the Demarara case which has been stained with hæmatoxylon and is exhibited under one of the microscopes on the table, will be seen to be composed of an accumulation of lymphoid cells covered by a thin epithelial layer. There are comparatively few bloodvessels to be found, and the tubercle presents an almost uniform appearance throughout, except for the scattered bands of connective tissue which divide it into poorly defined lobules. The lymphoid elements are situated in an alveolar network, and in many places giant cells may be observed. A very similar appearance is that of a transverse section made from the terminal phalanx of one of the fingers, in which even the bone has been destroyed by the disease. This preparation is also exhibited under one of the microscopes, and the uniform infiltration of all the tissues below the *rete Malpighii* will be noticed. Around the bloodvessels the cells are gathered in greater numbers, often forming concentric nest-like groups. The walls of the vessels are frequently thickened by the infiltration, and a partial obliteration of the lumen is often to be observed; the latter being caused by a proliferation of the endothelial lining. Other sections of the finger and of the nodule have been double-stained in order to show the *bacillus leprœ*. The method adopted was the well-known one of Ehrlich for staining tubercle bacilli; using either fuchsin or gentian-violet, followed in the first case by

methyl-blue or by sulph-indigotate of potash, in the second case by Bismarck-brown. The best results were obtained with fuchsin and methyl-blue. The bacilli are usually described as slender, rod-shaped bodies of a length equal to one-half or one-third the diameter of a red blood cell, often joined end to end in short chains or gathered in bundles, almost invariably lying in the great "lepra cells," and only rarely to be seen in the intercellular tissue. Under one of the microscopes I have placed a slide from the specimens of leprosy obtained in Vienna. This exhibits the bacilli as the usually accepted description states that they appear. Here the huge lepra cells crowded with the microbes are shown to perfection, and no scattered bacilli are to be seen. But in the Demarara specimens also shown this evening, the microörganisms are spread throughout the whole tissue, including even the epithelial layer, though here they occur but sparely. That they are to be found among the epithelial cells, and even in the hair follicles and in the hairs themselves, had been denied by most writers, but has since been affirmed by Unna (*Deutsche med. Zeitung;* Mitte December, 1885, 14) and later by Touton (*Virchow's Archiv*, civ. 381) and Neisser. You will be able to see them very distinctly among the epithelial cells in the specimens shown this evening. Elsewhere in the tissue they appear to a slight degree massed in regularly round or oval lepra cells, but chiefly free and either isolated or in irregularly shaped groups of various sizes. This, their occurrence to so great an extent free in the tissues, has been claimed by Unna (*loc. cit.*), but has been hotly contested by Hansen, Neisser, and Touton. If true, it removes one of the characteristic differences between the arrangement of the bacilli of lepra and those of tuberculosis. Unna declares that the lepra cell never contains a nucleus, and that it is, in fact, nothing more than an aggregation of bacilli held together by mucoid material. The appearance of the bacilli in the specimens from Demarara indicates the probable accuracy of this view. There is too much variation in the size, and too great an irregularity in the shape of the agglomerations, to warrant the belief that they are situated in a cell, and neither nucleus nor cell-wall can be detected.

Unna's method of preparation was somewhat peculiar. The sections, after being stained and cleared in oil, were dried with blotting paper and then *over the flame* until the oil was evaporated, and were then mounted in balsam. Other specimens were stained, then transferred directly from water to a cover-glass, warmed over the flame to absolute dryness and mounted in balsam; thus avoiding the use of oil. These methods are said to show the bacilli in unusual numbers. Hansen (*Virchow's Archiv*, ciii. 388) and Neisser (*Ibid.* 355) believe that Unna's

method destroys the cells, and that on this account, therefore, the bacilli have appeared to lie free in the tissue and lymph spaces. Hansen does not deny that they may lie in the lymph spaces, but thinks that when properly treated they will always be found *in cells* in these spaces. He brings forward other arguments to support his view, among others that the bacilli can never be found in the blood. Neisser makes the same statement. Kobner, however, declares positively (*Virchow's Archiv*, lxxxviii. 282) that they do occur both in the white blood-corpuscles and in the serum. He finds them free in the intercellular tissue as well as in lepra cells; but never in the epithelial layer.

Although not fully prepared to express an absolute conviction, yet the appearance of the specimens exhibited this evening makes me inclined to adopt the view of Unna with regard to the position of the bacilli in the tissue. I have avoided the special methods of preparation which he employed, and can see no reason why those which I have used should destroy the so-called lepra cells, if, indeed, these bodies really are of a cellular nature. It is a curious circumstance, too, that while the specimens from Vienna show bacilli in lepra cells alone, those from Demarara reveal scarcely any such structures, but are crowded with microbes lying free in the tissues. They have been prepared in precisely the same manner. Whether it is that these cells, or masses of mucus, whatever they may be, at some time in the course of the disease burst, or in some way become destroyed and spread the bacilli broadcast, I do not know, but it would seem at least possible. Dr. Lyell says (personal communication) that he easily finds the bacilli in the lymph from the leprous vesicles. This seems to be a proof that even if Hansen's view is correct regarding the position of the microbes in cells in the lymph spaces, these so-called cells must break down very readily and set the bacilli free. *January* 28, 1886.

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4. *Brain of a dement with numerous calcareous deposits scattered throughout both hemispheres and cerebellum.*

Exhibited by Dr. F. X. Dercum.

The patient was a woman, æt. about 45 or 50 years. She suffered from dementia interspersed with brief periods of excitement. The latter, however, did not differ from such as are frequently present in patients in whom no lesions similar to the ones about to be described are found. She also had phthisis.

The autopsy was held about seventeen hours after death. The calvarium was rather thicker than normal, though the dura presented nothing unusual. The pia and the arachnoid, however, presented opacities and thickening over the entire surface of the brain. The lateral ventricles were somewhat dilated. The velum interpositum was thickened, and the choroid plexuses pale and cystic. On making section of the brain the knife came in contact with hard calcareous particles scattered through the substance of the hemispheres; they varied in size from a quarter of an inch or more in diameter, to scarcely appreciable, sand-like fragments. They were largest and most numerous in the regions supplied by the nutrient branches of the Sylvian arteries—*i. e.*, the internal capsule, the head of the caudate nucleus, and the lenticular body. Toward the cortex they disappeared altogether. An examination of the cerebellum revealed similar bodies scattered in and about the dentate bodies of both lobes. Examination of the lungs revealed extensive tuberculosis, phthisis being the immediate cause of death. No calcification of the tubercles had taken place. Examination of the heart did not reveal any calcareous deposits either in the valves or great vessels. The kidneys were sclerosed.

A number of the calcareous particles found in the brain were removed from the tissue in which they were embedded and were then decalcified. Sections were cut and mounted as usual. The microscope revealed an enormous mass of irregular bodies, which were, for the most part, more or less cylindroid in shape. They were gnarled and twisted, and so densely packed that in some instances their nature could only be determined with difficulty. They proved to be vessels, the walls of which were much thickened, and in which extensive deposits of lime salts had taken place. In many instances the lumen of the vessels could readily be distinguished, in others again the lumen was entirely obliterated. In most cases it was exceedingly small. The process of calcification had, evidently, taken place throughout the walls of the vessels, though some of the smaller ones presented a mammillated appearance, as though the deposit had been in the form of an incrustation on the outer wall. The tissue between the vessels, and, also, that surrounding the nodules showed an extensive proliferation of nuclei. No undoubted evidence was present of any calcareous deposit in the neuroglia or in the true nervous elements. It is not improbable, however, that some of the smaller masses represented the calcified detritus of softened nerve tissue, but in almost every instance a more or less distinct vascular form could be made out. The most striking appearance under the microscope was the enormous number of the vessels, but at the periphery of a

nodule they would thin out, and frequently presented dilated lymph spaces.

The examination of the cortex also revealed a marked increase in the number of vessels and a general proliferation of the neuroglia. The most noteworthy point, however, was the shrunken appearance of many of the ganglion cells together with a great dilatation of the pericellular spaces.

Although calcareous deposits are known to take place in the falx, the tentorium, the dura, the pia, the main cerebral arteries, and even in the choroid plexuses, cases like the one described appear to be extremely rare. In 1850, Delacour described a case of imbecility in which small, hard, rough masses were found scattered throughout the centrum ovale. They were more especially noticed in the regions of the striated bodies, in the centre of each of which a hard substance the size of a bean was found. Delacour correctly interpreted these appearances as being due to the calcification, here and there, of the smaller vessels. The principal trunks were not affected. It is also of great interest to note that no calcareous deposits were observed in other portions of the body. This case, therefore, closely resembles the one before us.

In 1852, Rainy reported the case of a man who had died of inflammation of the hand "with purulent absorption." No symptoms of brain disease had been noticed during life, but on making sections of the brain, sharp spicula or bristle-like projections were discovered on the cut surfaces. They were evidently ossified vessels. The trunks of the smaller ones were of their natural size, but the minutest ramifications and the capillaries were much enlarged and altered in appearance, looking more like globules of oil joined together than like bloodvessels. This appearance, judging from Rainy's illustration, closely resembles that observed in some of the vessels in the present case—the mammillated appearance already mentioned.

Again, in 1855, Bamberger described the case of an epileptic dement, in which the smaller vessels of the brain were converted into a long, brittle framework, and which projected like so many wire points from the cut surface. Beside, in the corpora striata were found foci filled with yellow stony conglomerations.

In 1856, Virchow likewise described a case of dementia in which he found extensive calcification of the smaller vessels of the white matter of the brain. On section they stood out like bristles. Virchow explains this case as being one of metastatic deposit, there being marked atrophy of the cranial bone, as well as arthritis deformans.

In 1863, Marcé reported a case of complete dementia, in which the

brain had undergone much disintegration from bloody infiltration, and in which the cut surface felt rough, like the tongue of a cat. The calcareous deposits were, for the most part, aggregated and quite large. The capillaries appeared as though incrusted.

In 1866, Hubrich reported two cases, both dementia, in which bristle-like projections were noticed on the cut surfaces. They were found variously in the general white matter, the corpora striata, the optic thalami, cerebellum, etc. They could, in some instances, be drawn out of the tissue in which they were embedded, ranging at times from a half to three-fourths of an inch in length. Their nature was, of course, apparent.

Of the abóve cases, which are the only ones thus far reported, that of Delacour resembles most closely our own. In his specimen he found merely nodules of calcareous material. He makes no mention of the bristle-like appearance described by the other writers. This, it will be remembered, is also absent in the brain before us. Bamberger, on the other hand, records both lesions. He describes both the hard nodule and the bristle-like appearances. In the other cases, five in number, the nodules were absent, the bristle-like appearance alone being present.

Evidently, we have here two lesions which are closely allied. The difference between them we find to be more apparent than real, especially when we come to consider the probable method of their formation. Rokitansky, in his admirable *Lehrbuch der Pathologie*, 1856, vol. ii. p. 472, in giving a general description of calcareous deposits in the brain, based on Bamberger's case, explains them by supposing an encephalitis, in which the nervous elements disintegrate and become calcified; also that there is a sheath-like incrustation of the vessels. I believe this supposition to be, in the main, correct, and it accords most happily with what we know of the lesions of dementia in general. In the case just described, for instance, we have the general meningo-encephalitis so common in dements, and which is evidenced by the condition of the pia and arachnoid, and by the general increase of the neuroglia. The areas in which the concretions were found were probably foci of encephalitis of greater intensity than elsewhere. In these foci inflammatory changes in the walls of the vessels became pronounced, beside which, the vessels increased enormously in size and number; so marked is this increase, that these foci could with perfect propriety be called angiomata. At the same time, probably, the neuroglia underwent proliferation, and a corresponding destruction of nerve tissue ensued. Then, doubtless, the calcareous deposit took place. Why such deposit should have taken

place in the brain and not elsewhere, is, of course, a matter for speculation.

In regard to the five reported cases in which no nodules were found, the encephalitis, no doubt, lacked a focal character. The lime salts were simply deposited in a diffused manner in the inflamed walls of the arterioles and capillaries. *January* 28, 1886.

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5. *Medical Photography.*

Read by Dr. G. A. Piersol.

The character of photographical work which the medical man will probably desire to undertake may be divided into three groups: 1st. Photographing specimens; 2d. Photographing patients; 3d. Photographing microscopical specimens; to which, possibly, a fourth should be added—making lantern slides and enlargements, for the purpose of illustrating lectures.

As to outfit, all of these requirements must be borne in mind. At the present time, probably, the best equipment for medical work would be one of Walmsley's modified copying cameras, as manufactured by the American Optical Company, taking a 4¼ by 5½ plate, fitted with a first-class lens. Regarding the latter, those desiring the best, might limit their choice with advantage to the lenses made by Ross, Dallmeyer, or Beck. Personal experience with the Beck lens warrants a most favorable recommendation, as its superior qualities adapt it admirably to work requiring fine definition, with great focal depth. As a matter of economy, the excellent lenses of Darlot might be substituted for the higher priced English glasses.

As much less expensive, the "Waterbury" outfit (camera, lens, and tripod, costing but fourteen dollars) is worthy to be considered. The addition, however, of a light wooden extension is imperative to obtain sufficient length of bellows to reproduce specimens of normal size—a matter very often desirable. A lens adapted to medical work would be a rapid rectilinear combination of about 8½ or 9 inch equivalent focus—a shorter focus lens possessing insufficient focal depth.

In addition to the camera and lens, a simple dark-room outfit, embracing non-actinic lanterns, three trays, graduate, dust brush, light-proof box for opened packages of plates, and a few bottles, is necessary.

Proper illumination is essential for satisfactory results. An evenly

distributed toplight is most desirable. This can be obtained readily from a skylight; such a convenience, however, is frequently wanting. An excellent substitute may be easily had by removing the apparatus to the open air, and stretching over all a piece of muslin, thus providing a mellow, evenly distributed toplight, admirably adapted to photographing specimens.

In focussing, the use of a focussing-glass is advantageous. Focus without the diaphragm on a point lying midway between the extremes of the planes of the specimen; afterward put in the smallest stop (say No. 128, F = 45.2 English scale).

The small stop necessitates a rapid plate unless the exposure is unduly prolonged. A fast plate and small stop, when properly employed, yield pictures with the best possible definition, and great focal depth, qualities very essential to satisfactory representations of specimens. In order to insure acceptable results, a correct exposure should always be attempted.

The length of exposure depends upon many conditions. Among others, *actinic power of the light*, this being modified by sunshine or clouds, by time of day, and by season of the year; *color* of the specimen—a tissue deeply dyed with blood requiring much greater exposure than the alcohol bleached organ; *character of the sun;* size of *diaphragm;* sensitiveness of the *plate; strength of the developer.* Since these all modify the duration of any individual exposure, the oft-heard comparisons as to the bare number of seconds given upon various negatives are of very little importance. Each worker must determine for his own conditions the exact number of seconds required on the ordinary specimen, and, with this as a basis, changing conditions as occasion demands.

While there are many excellent formulæ, the following developer has always answered admirably, and with it, by slight modifications, all kinds of work may be satisfactorily performed:

No. 1.

℞.—Pyrogallic acid (Schering's)	1 oz.
Sodium sulphite	6 oz.
Water	48 f℥.

No. 2.

℞.—Sodium carbonate	4 oz.
Water	48 f℥.

No. 3.

℞.—Sodium bromide	1 ℥.
Water	1 f℥.

Use equal parts of No. 1 and No. 2, with three to five drops of No. 3 as a "normal" developer, the entire bulk of the developer being, say, three fluid-ounces. Remember, No. 2 accelerates, while No. 3 restrains development; No. 1 giving density.

A plate is fully developed when the deep shadows show some detail, and when the cardinal outlines of the picture show on the track of the plate with reflected light.

The most usual failures result from over-exposure and under-development. When a plate blackens rapidly, without the requisite amount of contrast between the high lights and deep shadows being at first present, we may strongly suspect over-exposure. Pour off the developer, flood with water, and then proceed once more with a developer, to which, say, twenty to thirty drops of No. 3 have been added.

The remedy for under-development is evident.

Strongly to be emphasized—select a good brand of plate, choose a reliable developer, and only change when there exists a strong reason for so doing; by this plan alone can the peculiarities and valuable qualities of any plate be learned. In addition to these fixed conditions, by at first employing but one lens and a single diaphragm, the path of the tyro is greatly smoothed.

In photographing a patient, if possible, a skylight should be used; where, however, none exists, we can fairly succeed in any well-lighted room, preferably one having two side windows. Opposite to window No. 1, and about four feet removed, we place our subject almost parallel —facing the second window. The lower half of window No. 1 is covered with muslin, while over the upper half is partly drawn the shade. Window No. 2 (furthest removed from the patient, but toward which he looks) is unobstructed. The camera is placed somewhere near the second window. Some simple background should be improvised, and the dark side of the subject should be lighted up by a reflector of white muslin. Since the actinic power of light is enormously reduced within doors, our exposure would be unduly prolonged if we employed the smaller diaphragms; we are, therefore, usually compelled to use the larger ones—No. 8 or 16.

Photographing microscopical specimens requires a camera with a long bellows—twenty-four to thirty inches. This may be had by adapting to the front of an ordinary camera an extension, which may be readily constructed of wood, card-board, or tin. The inner surface of the entire tract of the light from the objective should be lined with black paper with matt surface.

Any microscope admitting of a horizontally placed tube, with a good

stage and centring substage may be employed. Likewise any objective possessing good definition and flat field may be used. The eye-piece is best removed, the camera and microscope being united by simply inserting the draw tube into the extension of the camera, and wrapping the joint with a few turns of some black fabric.

Illumination is very important. Four sources may be considered: lamp, calcium, electric, and sunlight. Calcium and the arc electric lights, while quite satisfactory by way of experiment, are practically debarred by the expense and inconvenience attending their use. The incandescent electric light, at the present time, offers little advantage over good lamplight, and is far inferior to sunlight.

It may be stated once for all, that sunlight *properly* employed is the best possible illumination for photomicrography. As it requires some care in its employment, and, above all, the devotion of the busiest hours of the day, we may inquire what can really be accomplished by lamplight. Mature consideration and considerable experience justify us in saying that with care and proper manipulation, really good photographs may be made with powers up to the $\frac{1}{6}$; of course, very much higher lenses may be employed—even the $\frac{1}{15}$ and $\frac{1}{16}$—but thoroughly satisfactory lamplight work with powers approximating 1000 diameters is seldom, if ever, seen. One thing seems demonstrated beyond question, that with any power it is almost impossible to obtain the soft, yet brilliant micro-negative—full of detail and vigor—that sunlight is capable of yielding.

For lamplight, a lamp with a single moderately broad wick should be employed—the edge of the flame being turned toward the object. For very low powers, 3 inches to $1\frac{1}{2}$ inch, no condensing lens is necessary. With higher powers, the interposition of a 4 inch focus plano-convex lens is advantageous, and with still higher lenses (say from the $\frac{2}{3}$ up), some form of substage condenser, in addition, is almost imperative. After many experiments with various forms, a "B" eye-piece as a condenser seems to yield the best results, giving a powerful and evenly distributed illumination. Accurate centring and even distribution are of the utmost importance. First centre the substage condenser, then interpose the bull's eye, and centre again without disturbing the substage condenser. Avoid "pushing" the illumination, as when the condenser is racked too high the margin of the field is distorted.

Experience teaches the advisability of using a slow, "clean working" plate (say Carbutt's "B"), in preference to the very rapid brands. A mirror placed at the end of the work-table, opposite the ground-glass of the camera, is of great assistance in adjusting illumination; a small hand-glass is also convenient to reflect the picture when hunting fields.

The most important, as well as difficult, manipulation is focussing. With a short "pull" no especial requirements, but when a long camera is extended, removing ground-glass to three or four feet, some arrangement of moving adjustments is necessary.

The simple arrangement with cord and weights, which we devised eight years ago, and which, of late, has become extensively used, we still recommend as meeting all possible requirements. It has been put to the severest test with powers of over three thousand diameters, and never has been found wanting, either in delicacy or accuracy. A modification may be applied to the coarse adjustment.

A focussing-glass is indispensable; the point of most accurate focus is where the image seems to melt into the ground-glass. Preparations must be well differentiated and thin, possessing sufficient contrast and actinic opacity. In certain cases, by using glass of complementary color to stain, excellent results are obtained.

While lamplight suffices for low powers, yet, for the high powers, and for difficult work, such as bacteria, sunlight, by all odds, is to be preferred. By arranging a simple mirror, to swing laterally and vertically, we can substitute the necessity for a heliostat. Light from this mirror passes through a plano-convex lens of 8 inch focus, through a cell of ammonio-sulphate of copper to the substage condenser. Here, again, accurate centring and evenly distributed illumination are absolutely essential for good results. To those aspiring to the best results, especially with difficult subjects and high powers, a trial of sunlight is strongly urged, as by this illumination alone are many of the capabilities of photo-micrography rendered possible.

[Succeeding his address, the lecturer exhibited on the screen about fifty examples of various kinds of work. Nearly half of these were photographs of pathological specimens; the remainder were photo-micrographs of histological and pathological preparations, taken with objectives giving from the lowest to the highest amplifications, among which an admirable photograph of the *bacillus tuberculosis* attracted much attention.] *April* 22, 1886.

6. *Specimen from a case of tuberculosis of both lungs, with implication of the suprarenal bodies and tuberculous ulceration of the colon—symptoms of Addison's disease.*

Exhibited by Drs. J. C. Wilson and W. Osler.

The specimens exhibited were removed from the body of a gentleman 38 years of age, married, and by occupation a physician. Family history bad; his mother, his paternal grandfather, and two uncles, having died of pulmonary consumption. The patient's health was fairly good until about two years ago, when he had well-characterized mild enteric fever, from which he made what was regarded by his physician as a good recovery. He did not, however, fully regain his strength, and an occasional cough, from which he had suffered for some years, began to be persistent and annoying. He was able, however, to resume his usual occupation. During the spring and summer of 1885 he began to lose flesh and strength, his appetite became irregular and capricious, his cough more troublesome. The cough was especially annoying on assuming a recumbent position and during the early hours of the night. Expectoration was rare and always mucoid in character. He suffered from occasional irregular chills which were regarded as malarial; night sweats from time to time annoyed him. After a cold contracted while driving in September, all the symptoms were aggravated and the patient began to experience more or less constant deep pain in the epigastric region. This pain he described as of a tearing or dragging kind not aggravated by food and liable to paroxysmal exacerbations of considerable intensity. He now noticed that his complexion, always dark, was becoming deeply and somewhat irregularly pigmented. About this time he began to suffer from occasional vomiting, for which no cause could be ascribed. At intervals of five or six days the contents of the stomach were thrown up without pain or distress, and equally without relief to the ordinary symptoms of his malady. The bowels were moved with remarkable regularity once a day, the evacuations being fluid and containing small sponge-like masses. The urine, upon repeated examination, yielded no trace either of albumin or sugar. Notwithstanding his growing weakness and sufferings, the patient delivered a course of lectures in the institution with which he was connected during the winter, and devoted some hours each day to literary work in addition to the discharge of other professional duties. In March, however, he completely broke down and came to this city for treatment. The appearance of the patient,

when he first came under observation here, was that of well-marked Addison's disease. The face was deeply pigmented, the discoloration being more marked about the brows, eyelids, and lips. The mucous membrane of the mouth was but slightly affected, the hands were much discolored, the finger-tips clubbed, the nails incurvated, the body and limbs were also much discolored, especially about the flexures. The pigmentation over the site of a former blister and in the neighborhood of an old injury near the left knee, was peculiarly intense.

Anæmia was of moderate degree, the number of red corpuscles, as counted by Dr. Osler, being 4,200,000.

The patient suffered from a sense of great weakness, which was increased and accompanied by dyspnœa and faintness upon exertion. His mental condition was clear.

The clinical phenomena relating to the digestive system remained the same, with the exception that the appetite curiously increased during the last weeks of the patient's life.

The pulmonary symptoms grew rapidly worse. Physical examination revealed dulness over the upper lobe of both lungs, more marked and more extended on the left side. There were also feeble bronchial respiration and occasional crepitant and subcrepitant râles.

The heart's action was regular, 90 – 110 per minute, very feeble; there were no adventitious sounds.

The area of liver dulness was somewhat increased; that of splenic dulness was normal.

The abdomen was neither distended nor retracted; it was resonant upon percussion, with the exception of a limited area in the left iliac region, where there was diminished tympany and some obscure resistance on palpation. These signs were afterward found to correspond to thickening of the colon and enlargement of the glandulæ epiploicæ in the region of an ulcer.

There was slight epigastric tenderness upon pressure. Death took place May 22, 1886.

Autopsy, thirty-six hours after death.—Moderate emaciation. Pigmentation not so deep on trunk as it was some weeks before death; on face quite as intense.

In *abdomen*, no chronic peritonitis; membrane not dark colored. In the course of the colon were several thickened and congested portions, one of which, about the middle of the sigmoid flexure, was very firm, and the appendices epiploicæ, and adjacent mesocolon were greatly enlarged. There were several calcified lymph glands in the mesentery. Branches of portal system moderately full.

In *thorax* universal adhesions on right side; on left, at apex and postero-lateral regions.

Heart of medium size; chambers contained dark clots. Valves normal. Muscle pale, and showed fatty changes.

Lungs: Upper lobes extensively diseased, and in great part airless. Section showed many groups of tubercles in a fibroid and pigmented tissue. Here and there a small caseous nodule. No cavities. The smaller bronchi a little dilated. In the left lung, one of the large bronchi, passing to the apex, was plugged with firm, cheesy matter. Between the groups of indurated tubercles and at the margins the tissue was emphysematous. The process was most advanced in the left lung. In the middle lobe of the right and in the lower lobes there were a few groups of firm tubercle.

Spleen of normal size, and presented scattered cheesy tubercles, with firm capsules.

Stomach: Extensive post-mortem solution at cardiac end and in œsophagus. Mucous membrane pale; veins full.

Pancreas healthy.

Duodenum normal. Bile-ducts pervious.

Small intestine presented a few small tubercles in Peyer's patches, but no loss of substance. In large intestine five large areas of ulceration, evidently of long standing as the bases of the ulcers, were cicatrized, and in one or two the calibre of the bowel was reduced.

Kidneys, suprarenals, and aorta removed together for dissection. The tissue between the cœliac axis and the head of the pancreas was puckered by the presence of three or four calcified lymph glands. The left splanchnic nerve entered a large, normal-looking semilunar ganglion, circular in form, which embraced a small accessory suprarenal about the size of a pea. The right splanchnic passed to a smaller ganglion, which also looked normal. There was no "matting" or thickening about these bodies, and the nerves passing from them could be readily traced. Just to the right of the cœliac axis the tissues were a little matted. The suprarenal capsules were small and flat, and had lost the normal shape. The left seemed lower than usual, and was separated by a distinct interval from the kidney. It presented two firm cheesy masses which occupied the greater portion of the gland. In the thinner, peripheral parts remnants of cortical substance could be seen as small islands of yellow-brown tissue imbedded in a gray fibrous stroma. The right gland was flatter and thinner, puckered on the surface, and presented no trace of normal tissue. A large, flat, cheesy mass occupied the greater part of the organ. The kidneys were large but normal; veins full. Micro-

scopical examination of the capsules showed tubercle bacilli in scrapings of the cut surface. The section presents the usual character of the fibro-caseous change so often described in these glands. There was also marked atrophy of the cortical regions, due apparently to an increase in the interstitial tissue. The nerves passing to the glands and the semi-lunar ganglia presented no essential changes. There was the usual pigmentation of the nerve cells. *May* 27, 1886

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7. *Address of the retiring President, Dr. J. C. Wilson.*

Delivered October 14, 1886.

It is my agreeable duty, in accordance with the By-laws, at this the annual meeting in October, to revise the work of the Society for the past year, and make such suggestions in regard to its labors and the mode of conducting them as may occur to me.

This requirement, which has been faithfully carried out by my predecessors, since it went into effect fifteen years ago, is an excellent one. It has something of the importance of the annual stock-taking and balance sheet of a business enterprise. It enables the Society to determine its actual condition as a working body, and to compare that condition with the past at fixed periods; to estimate its progress; to observe its shortcomings; and it brings into clearer light the causes of both.

Perhaps a somewhat formal address from the Chair at the end of the year may suggest to each individual member questions in regard to his contributions and gains in the Society's work, which he can best answer for himself. Certainly, it brings home to me a strong sense of the responsibilities which attend the honor you have conferred upon me. It is not a very difficult task to discharge the mere parliamentary duties of this office when, as here, good-will prevails and unselfish devotion to science unites all interests in efforts to further the common cause. But to sustain the interest in the Society's work among members forgetful of it in the press of other affairs, to remind the old members of its scientific and financial needs, to see that the active members comply with the regulations concerning written reports of the specimens which they exhibit, to call forth and encourage active and apposite discussion, to aid the various committees in their labors, and to see to it that the valuable records of the Society's work embodied in the as yet unbroken series of the transactions is continued without interruption, are duties which call

for earnest and devoted application on the part of your presiding officer. Within the compass of my ability I have sought to discharge these duties faithfully, yet I feel that the success which has crowned my efforts is due rather to your hearty coöperation than to any effort of mine.

The Society numbers upon its roll of active members at the present time 162, of whom 10 were admitted to membership during the past year. The corresponding members are 11, none having been added during the year.

The meetings have been well attended, the maximum number present at any meeting being 32, the minimum 12, and the average over 20. At no meeting did the Society lack a quorum for the transaction of its affairs, nor was there any meeting without the exhibition of valuable specimens and reports of interesting cases.

Dr. J. Collins Warren, of Boston, delivered the address at the semi-annual meeting in October, and Dr. Pearsol, of this city, that in April. Both these meetings were largely attended.

The Recorder informs me that there were presented during the year no less than 151 specimens or series of specimens. Many of these were of extreme interest, by reason of their rareness; others were of value as shedding light upon obscure questions in pathology; several were exceedingly instructive as illustrations of variations from the usual anatomical changes in morbid conditions, while a few were well worked up examples of familiar lesions. The series of specimens presented by some of the members have been of great value and have elicited interesting discussions. I would suggest that this method of illustrating special morbid processes might be extended with profit. In some cases two or more members might combine in the exhibition of specimens and in remarks upon the pathology of a given disease in such manner as to make it the subject of the meeting. Combinations of this kind would often be peculiarly favorable for the exhibition of specimens and observations by members engaged in special work.

Of the 151 single specimens or series, thirty-two were "card specimens." This plan of displaying certain specimens of general rather than particular interest, is capable of being made much more prominent at our meetings. A table for this purpose placed at a more accessible position at the back part of the hall near the entrance, would be an improvement upon the plan hitherto employed.

Where so much excellent work has been done, it is not easy, nor would it be proper for me to dwell at length on the labors of individual members. I cannot, however, refrain from grateful allusion to the unflagging enthusiasm which has made so much that is of value among the rich

pathological material of the Coroner's office available for your study. Other members of the Society have in the past served as Coroner's physicians, but never before in our history has the material of that office been utilized for scientific purposes here to the extent which has been made possible by the efforts of Dr. Formad.

The discussions have been, as a rule, interesting and instructive. Too often, however, important specimens have failed to call forth remarks from members whose opportunities have especially fitted them to speak with precision and authority upon points in pathology not always familiar to their fellows in the Society. This is not right. The purposes of our Society demand full and frank discussion. Debate is suggestive; it widens knowledge; it corrects error. It gives all-around views of subjects. Even the flint and steel of difference of opinion strike useful sparks. *E scintilla lux.*

The rule of the Society which requires every member presenting a specimen to furnish the Recorder with a written description of the same has been violated in no less than forty instances during the year. This circumstance is of much more serious injury to the interests of our Society than many of you seem to suspect. It occasionally happens that in order to present a recent specimen in its fresh condition, the rule must be set aside on account of the lack of time needed to prepare a paper. But in such instances permission should be invariably obtained by a formal vote of the Society. On all other occasions, a concise written description of the specimen ought to be furnished for the records and for the published proceedings in the journals. The neglect of this regulation deprives the Society of a fair presentation of its whole work, both in the journals and in its transactions.

I trust that in thus directing the attention of the members to the excellent by-laws on this subject, all that is necessary to secure its observance in the future has been done.

In fifteen instances members presenting papers have asked for, and obtained the permission of the Society to publish them separately. These communications remain, as a matter of fact, the property of the Society, and will be reprinted in the appropriate volume of the *Transactions.*

It is my good fortune to be able to announce that the delay in the publication of the twelfth volume of the *Transactions*, due to lack of funds, is now, owing to the generosity of several friends of the Society, practically drawn to a close, a sum sufficient to warrant the Publication Committee in beginning to print that volume, being now subscribed for that purpose. It is a subject of congratulation that there is to be no interruption in the

long series of these volumes, which embody the records of the scientific work of the Society since 1857, and which have now a historical, as well as a scientific value.

A number of important changes in the by-laws have been made in the course of the six years that have elapsed since the last copies were issued. I would, in view of this fact, suggest that a Committee be appointed to revise the by-laws, and that when the Society shall have acted upon such revision, the Publication Committee be directed to have a new edition printed for distribution among the members.

With this meeting the Pathological Society begins its thirtieth year. I will not here enter upon any account of its history. In an admirable address delivered by one of my distinguished predecessors, Dr. James H. Hutchinson, upon retiring from the Chair in 1872, and printed in the fourth volume of the *Transactions*, the history of the Society to that date is narrated in an interesting manner. To do this, no one better qualified than Dr. Hutchinson could have been selected He was one of the twenty-seven original members of the Society, and for many years its Secretary. To his interest in the welfare and purposes of the Society, and that of a very small number of others, is wholly due its continued existence during the troubled years of the War of the Rebellion. It is stated that, prior to 1872, this was regarded as the best-working medical society in the city.

Since the date of that address, the career of the organization has been one of almost uneventful usefulness and prosperity.

For the most part, the older members have drifted out of its active work; from time to time resignations have taken place; several names have even been dropped from the roll for the non-payment of dues. Many honored members having finished their course here have gone over to the majority. But new and young life steps into the vacant places, and the mutual labor of learning and teaching goes on. And may it long go on! For the work done here is work done for the work's sake—the best kind, I had almost said the only true kind of scientific work. It is hard work, time-consuming, often performed with great sacrifice of personal ease, always without recompense except that which comes with knowledge, but it is work that is well repaid.

It is with you to sustain our old reputation as the best working of the local medical societies.

I cannot close these brief remarks without expressing my earnest thanks for the uniform consideration I have received at your hands in the discharge of my official duties, which have been thus rendered at once easy and pleasant.

8. *The hæmatozoa of malaria.*

Read by Dr. W. Osler.

Our knowledge of the animal parasites infesting the blood has been of late enriched by observations which show that certain of these hæmatozoa, as they are called, are more widely distributed and more important than we had hitherto supposed. Parasites belonging to the sporozoa, and to the nematode and trematode worms, have long been known to occur in the blood of various animals. Recent investigations prove that the flagellate protozoa are also not uncommon blood parasites, and it is possible that they may be the pathogenic organisms of certain diseases. I propose in this communication to give an account of the hæmatozoa which have been found in persons suffering with the various forms of malaria.

Historical.—Our knowledge of the blood-changes I am about to describe, dates from the researches of Laveran, in Algiers, which were communicated to the Paris Academy of Medicine in 1881 and 1882, and which were finally embodied in a large work on the malarial fevers, published in 1884.[1] He found, as characteristic elements in the blood of persons attacked with malaria, (1) crescentic pigmented bodies; (2) pigmented bodies in the interior of the red corpuscles, which underwent changes in form, described as amœboid; and (3) a pigmented flagellate organism. These forms were looked upon as phases in the development of an infusorial organism which he regarded as the germ of the disease. Richard[2] confirmed these observations. A more general interest in the question was aroused by the publications of Marchiafava and Celli,[3] who found in the blood of malarial patients at Rome the bodies described by Laveran. They figured carefully the alterations of the organism in the interior of the red corpuscles to which they gave the name Plasmodium malariæ. Councilman, of Baltimore, has more recently confirmed these observations.[4] The pigment granules so numerous in the interior of the red corpuscles in cases of "comatose pernicious fever," which appear to be included in a hyaline mass, are, according to Marchiafava and Celli, and Councilman (who had previously described them[5]), these amœboid parasites deeply laden with altered hæmoglobin.

[1] Traité des Fièvres Palustres, Paris, 1884.

[2] Comptes Rendus, 1882.

[3] Fortschritte der Medicin, Nos. 14 and 24, 1885.

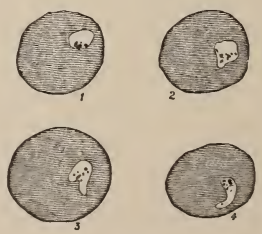
[4] Paper read before the Association of American Physicians, June, 1886.

[5] Councilman and Abbot: American Journal of the Medical Sciences, April, 1885.

Technical details.—The finger pad from which the blood drop is taken should be thoroughly cleansed, and if the examination is made during a paroxysm, the sweat which may exude after the friction and drying should be removed. Attention to these, apparently trivial, details will secure specimens of blood free from small particles of dirt, and facilitate considerably the search for pigmented bodies. The layer of blood beneath the top cover should be very thin and uniform, the corpuscles, as far as possible, isolated and not aggregated in clumps or in rouleaux. It is well to surround the cover with paraffin if the examination is prolonged. No reagent of any kind should be added. Cover-glass preparations may be made and stained in methyl-blue or fuchsin, and mounted in balsam. Osmic acid preparations may also be employed. Although these bodies may be seen with a power of 500 or 600 diameters, it is essential for the satisfactory study of the changes to use higher powers. I have uniformly worked with the $\frac{1}{12}$ homo. immersion of Zeiss, and the $\frac{1}{15}$ im. of Reichert. Stricker's warm stage will be found useful.

Description of the Bodies. 1. *The forms which exist within the red corpuscle.*—(*a*) The most common alteration in the blood of malarial patients is presented by a pigmented structure inside the red corpuscle. The attention of the observer will most likely be first attracted by the presence of a few dark grains in the stroma, and a careful study of a suitable specimen will soon lead to the conviction that these are not scattered loosely, but are enclosed in a finely granular or hyaline body in the interior of the corpuscle (Fig. 1). The red disks in which they

Fig. 1.

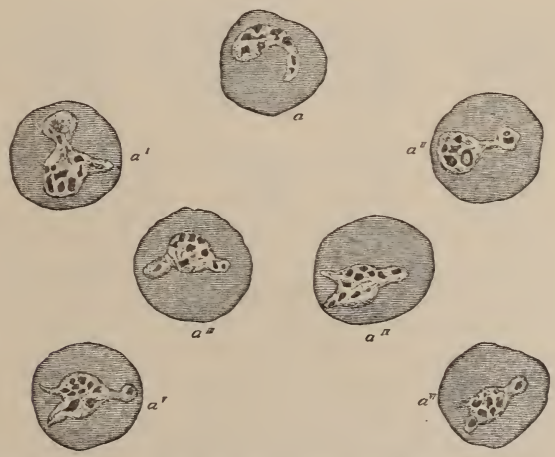


Amœboid body in red blood-corpuscles. The sketches were made at intervals of five minutes.

occur are usually larger, look flat, and are very often paler than normal; they may, indeed, exist only as colorless shells. The number of corpuscles so affected varies extremely in different cases. In some instances they are readily found after a search of a moment or two, but, in other cases, a prolonged examination may be necessary. Only one is usually present

in each corpuscle, but two or three, or even four, may occupy the stroma. They vary greatly in size, the smaller ones not occupying a fourth of the corpuscle, while the larger ones may almost fill it. A delicate contour line can usually be seen separating the body from the stroma; at times this is very indistinct, particularly if the illumination is very bright. The substance appears hyaline, or very finely granular, and the pigment grains are scattered irregularly in it. They may be very numerous, and give a dark aspect to the body, or they may be scanty. They frequently present rapid Brownian movements. Occasionally a vacuole may be seen in the interior of the body. In several instances the bodies appeared to be enclosed in a clear space—vacuole—in the stroma. When first seen they are more or less spherical, but, as already stated, the outline may be indistinct. The pigment granules may be seen to alter their position in relation to each other. If the margin of

Fig. 2.

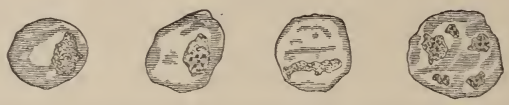


Case VI. Pigmented body in red blood-corpuscle; outlined with camera (1–12 Zeiss, C eye-piece), by Dr. J. P. C. Griffith; illustrating some of the changes during an hour and a half's observation. *a*, at 11.45, slow alterations in outline, and the pigment-granules are in active dancing motion. *a*I, 12.15. *a*II, 12 25, body has rotated as well as altered its shape. *a*III, 12.30. *a*IV, 12 40. *a*V, 1 o'clock. *a*VI, 1.02.

the body is carefully observed, slow changes can be seen, which gradually bring about alterations in shape. These movements, which appear to be amœboid in character, can often be traced with great ease. They are well represented in Fig. 1, and, better still, in Fig. 2. Changes in position of the body of the corpuscle result from them. They are decidedly slower than the amœboid movement of the colorless corpuscle.

I have not seen any evidence of migration from the corpuscle. In dry preparations these bodies stain deeply with gentian violet or fuchsin and present a granular stroma, in which the pigment grains are imbedded. (Fig. 3.)

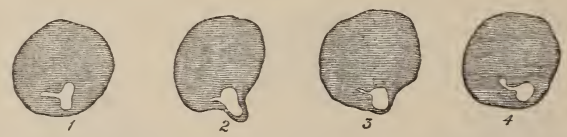
Fig. 3.



Cover-glass preparation of blood stained with fuchsin. The amœboid bodies stain deeply in the corpuscles. Some of them are not pigmented.

(*b*) In seven cases peculiar hyaline structures existed in the interior of the red corpuscles, which differ from the bodies just described, in the absence of pigment and in the much greater activity of the changes. Fig. 4 illustrates the appearance and the alterations in outline. These

Fig. 4.



Sketches of the alteration in form of one of the hyaline bodies; 1, at 7.8 P.M.; 2, at 7.12; 3, at 7.15; 4, at 7.20.

bodies are devoid of structure, and the corpuscles in which they are present are not so pale as those with the pigmented forms. Marchiafava and Celli, who have given an excellent plate of these bodies,[1] regard them as the initial forms of the pigmented bodies. One does occasionally see appearances indicative of commencing pigmentation, but they have not, as a rule, the solid aspect of the pigmented bodies. In three cases I have seen the following remarkable changes. The hyaline body, while actively changing shape, suddenly burst from the stroma, and disappeared, or formed only a few granules. Thus, in a red corpuscle, there were, at 3.40 P.M., two hyaline, irregular-shaped bodies, which were changing rapidly in outline. The alterations were so marked that the physicians present at the time had no difficulty in seeing them. The stroma of the corpuscle was of full color. At 3.50 P.M., as I was carefully watching these forms, the corpuscle suddenly ruptured, and gave exit to two distinct masses, which quickly broke up into ten or twelve spherical bodies. No change took place in these after twelve hours,

[1] Fortschritte der Medicin, No. 24, 1885.

except that they became pale and indistinct. The stroma of the corpuscle became quite colorless. On two other occasions a similar phenomenon was witnessed, but in one no trace could be seen of the extruded material. This is evidently a physical change, and I think these very pale hyaline bodies must be carefully distinguished from the pigmented forms, though possibly associated with their early development.

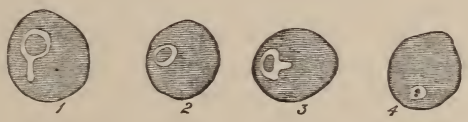
(*c*) In seven cases there were vacuoles in the red corpuscles containing solid-looking bodies of various sizes and shapes. Certain of these structures resembled micrococci very closely (Fig. 5), and stained deeply in

FIG. 5.

Cover-glass preparation, showing corpuscle with solid, deeply stained bodies in small vacuoles.

aniline dyes; but others, often in the same corpuscle, were larger, more irregular, and altogether different in appearance (Fig. 6). The smaller

FIG. 6.



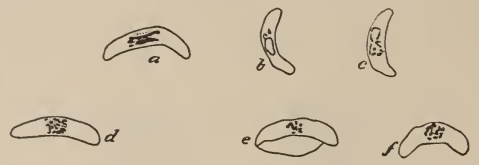
1, 2, 3, larger solid bodies in the interior of vacuoles (?) which alter in outline. 4, a red corpuscle, with a small vacuole containing small pigment-granules of a deep brown-red color.

ones were usually highly refractile, and, when two were together, the appearance suggested a diplococcus. In three instances these bodies had a deep brown tint, as if composed of pigment. The larger bodies were homogeneous, very variable in size and shape. No movement was noticed in them, but the outlines of the spaces in which they lay sometimes changed actively. In Case XXIX. these bodies were very abundant, and for days formed the only noticeable alteration in the corpuscles.

2. *The free forms.*—(*a*) Pigmented crescents. These bodies, which were found in eighteen cases, present remarkable features in appearance and structure. The form was usually that of a beautiful crescent (Fig. 7), with rounded or gently tapering ends; but the degree of curvature was variable, and many forms were almost straight. The length is about double that of the width of a red corpuscle, sometimes more. They are

not attached, and they never show any motion. Joining the ends of the crescents—or, more correctly, at a little distance from the points—a narrow line can often be seen on the concave margin (Fig. 7, *e*). The body of the crescent appears made up of a structureless, homogeneous material, in the centre of which is a prominent collection of pigment granules. This, with the peculiar form, makes these bodies very easily

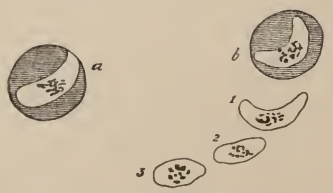
Fig. 7.



Crescents, *a*, *b*, *c*, show the slow alterations in the form of the pigment, as sketched at 9.20, 10.40, and 10.55 A.M. *e*, shows the narrow membrane sometimes present in the concave side.

recognizable in the blood, even when closely surrounded by the corpuscles. The pigment is very dark in color, distinctly granular, and varies somewhat in its arrangement. As a rule, it is central and aggregated, either in a heap, or assumes the form of a band placed transversely to the axis of the crescent. In some instances it is more scattered, but I have never seen it at either end of the body. Although the most careful examination fails to detect any movement in the hyaline substance of the crescent, yet the existence of such may be inferred from the very positive movement which the pigment granules undergo. Fig. 7, *a*, *b*, *c*, represents these alterations; changes in form are exceptionally seen, as shown in Fig. 8 (1, 2, 3). A crescent became within an hour, an ovoid

Fig. 8.



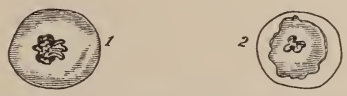
a and *b* show crescents in the interior of red corpuscles; 1, 2, and 3, changes in a crescent.

body. Sketch 1 was made at 9.40, 2 at 10.10, and 3 at 10.30 P.M. The outline of these bodies is very clear and defined. Ovoid, elongated, and rounded forms of identical structure are also met with, but the crescents predominate. The number is variable, from one or two in a slide, to six or eight in the field of the 1–12th im. Though almost

always free, they occur sometimes in the interior of a corpuscle, indicating, doubtless, the mode of development (Fig. 8, *a* and *b*).

(*b*) *The rosette form.*—In six instances there were rounded bodies, a little larger than red corpuscles, with a dimly granular protoplasm, and in the centre a rosette of pigment (Fig. 9). Some of these appeared to be enclosed in a delicate membrane, others were free. In six cases

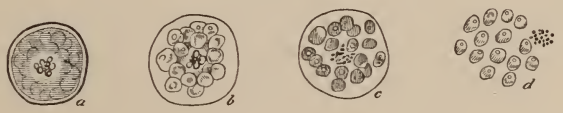
FIG. 9.



Rosette form : 1 free ; 2 within the shell of a red corpuscle.

remarkable changes were seen in these forms, of the nature of segmentation. Thus Fig. 10, *a*, represents one of these as seen at 6 P. M., Septem-

FIG. 10.



Segmentation of a rosette form : *a* at 6 P.M. ; *b* at 6.10, segmentation proceeding ; *c* 6.30, segmentation complete ; *d* 7.40, small free bodies.

ber 4th. At 6.10 (*b*) there were distinct indications of segmentation in the finely granular protoplasm. At 6.30 (*c*) this had resulted in the formation of twelve or fifteen rounded bodies clustered about the central pigment, and still enclosed in the sheath. At 7.40 (*d*) the shell had burst, and given exit to the small corpuscles, which presented a tiny speck at or about the centre. At 10.40 they had not undergone any material change. In Case LX., one of quartan ague, this phenomenon was repeatedly observed. The development of the rosette form can, I think, be traced from the intracellular pigmented bodies, which increase in size until the entire corpuscle is filled. In some instances the body was surrounded by the remnant of the red corpuscle, in others there was no trace of it. The pigment granules gradually collect in the centre of the body in a more or less distinct rosette. I thought these changes had been overlooked by the writers on this subject, but I find that Golgi[1] has given a very full description of them, and has beautifully figured

[1] Sulla Infezione Malarica, Archivio per le Scienze Mediche, vol. x. No. 4, 1886.

the development of the rosette form from the intra-cellular pigmented bodies. He has followed the process of segmentation with much greater detail than I have been able to do.

(*c*) *Flagellate organisms.*—Two or three years ago, when I first read Laveran's papers, nothing excited my incredulity more than his description of the ciliated bodies. It seemed so improbable, and so contrary to all past experience, that flagellate organisms should occur in the blood. The work of the past six months has taught me a lesson on the folly of a scepticism based on theoretical conceptions, and of preconceived notions drawn from a limited experience. Flagellate bodies were seen in seven cases, never in great numbers, usually only one or two in a slide. They are smaller than red blood-corpuscles, often not more than half the size. A specimen in one case was equal in one diameter to a red corpuscle lying near it. They are round, ovoid, or pear-shaped; the protoplasm finely granular, and in every instance contained pigment, usually central, which often displayed rapid Brownian movements (Fig. 11). The

FIG. 11.

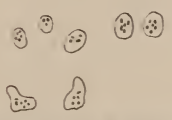
Flagellate forms.

flagella are variable in number; one, three, and four were noted in different specimens. The length, as closely as could be estimated, was two or three times that of the body. They are exceedingly delicate, gently tapering, and, except in one instance, I could not determine the existence of a small terminal knob, figured by Laveran. The movement is exceedingly active, and the lashing of the long filaments may be sufficiently strong to drive away the corpuscles in the vicinity. The undulatory movement caused by the play of the filament over the surface of a group of corpuscles may attract the attention of the observer before he sees the cilia. The motion does not persist long; in none of the specimens which I examined, for more than half an hour. In one instance, the flagella disappeared in the short interval between two observations, but I could not determine what became of them. I have not seen the free-swimming cilia described by Laveran, but Dr. Councilman tells me that he has confirmed this observation. I have not been able to discover

either nucleus or vacuoles in the flagellate organism. Slight, irregular changes in outline occur, due to slow movements in the protoplasm.

(*d*) Small round, pigmented bodies, from one-fourth to one-half the size of a red corpuscle, were not uncommon in some cases (Fig. 12).

Fig. 12.



Small free pigmented bodies, some of which show amœboid movements.

Usually, they remained unchanged, but in several instances they showed amœboid movements. The smaller ones about equal in size the products of subdivision of the rosette form.

Before proceeding to discuss the nature of these bodies and their relation, I will briefly refer to the condition of the blood corpuscles.

The red corpuscles showed no other notable alteration save that already described. The pigmented organism evidently destroys the vitality, and consumes the hæmoglobin, for the affected cells become pale, often spherical, and, finally, are reduced to the condition of mere shells; except in cases of pronounced anæmia, the variations of the corpuscles in size and outline were not great. The colorless corpuscles were in some cases increased in number, and in very many instances contained dark granules. In several specimens they were observed to contain the pigmented organisms. In Case XL., a crescent had been included (Fig. 13), and, in Case LI., the process of inclusion of two free pigmented bodies was

Fig. 13.

A colorless corpuscle containing a crescent.

watched during half an hour. The blood plates were, as a rule, scanty, even when the anæmia was pronounced. No pigment was seen in them.

Types of malaria studied.—Of the seventy cases examined, a majority were instances of ordinary intermittent fever, chiefly quotidian and tertian, with two quartan cases. There was one case of remittent fever, one

of comatose pernicious malarial fever, and the remainder were cases of malarial cachexia or chronic paludism, with occasional outbreaks of fever, with or without chills. In all of the cases, with the exception of seven, one or other of the forms above described was found in the blood.

Relation of the forms to the varieties of malaria.—The pigmented amœboid bodies are met with in acute and chronic cases, but they may be said to be specially characteristic of the more acute manifestation of the disease. In recent examples of quotidian or tertian ague which had not been under treatment, the amœboid intracellular forms were almost invariably present. I will refer subsequently in detail to the cases in which they were not found. The hyaline non-pigmented forms, and the vacuoles containing solid bodies, also occur in the acute cases; indeed, these latter forms were the only alterations noted in several instances. Thus, in Case XXIX., a man aged 48 was admitted to the Philadelphia Hospital September 27th, in a chill. He had had a paroxysm ten days before, and had suffered with malaria several years previously. The blood examined during the hot stage showed no pigmented bodies, but numerous corpuscles containing the vacuoles shown in Figs. 5 and 6. The chills occurred on the 28th, 29th, 30th, and October 1st; and each day the blood was carefully examined, without finding other bodies than those in the vacuoles or hyaline spaces. On October 1st, the patient began with ten grains of quinine twice a day, which was continued for five days. He had no chill after October 1st. On the 7th there were crescents in tolerable numbers, which persisted until the 27th, the date of the last examination.

The crescents appear to be associated with the more chronic forms of malaria, or with acute cases which have been under treatment for some time. Of eighteen cases in which they were present, in twelve there was a history of infection lasting from six weeks to six months. In many, the cachexia was marked, and the spleen greatly enlarged. In six instances the attacks were recent—under a month; but in every one of these cases quinine had been taken. As a rule, the crescents occurred alone in the blood; but there were cases observed in which the pigmented amœboid bodies, the rosette form, and the flagellate organisms, were also present. I did not find the crescents in any recent cases of intermittent fever which had not been under treatment.

The rosette form, with its peculiar segmentation, occurred in six cases, and always in association with the amœboid intracellular bodies. Case XXXI.: tertian ague, examined in fourth attack; no medicine. Case XXXIII.: quotidian for seven days. Case XXXVII.: quotidian for six weeks, anæmic, and had a large spleen; took quinine one day. Case

XXXIX.: quotidian for seven days. Case LVIII.: quotidian, on and off, for six weeks, then stopped; now daily chill for a week. Case LX.: quartan for three weeks. I have noted these details, as this form has not been much studied, and as Golgi seems inclined to ascribe to it an important connection with the development of the paroxysm. It was only observed in acute cases which had not been under treatment.

The flagellate organisms were present in seven cases, six of which were chronic forms, and one an acute case of three weeks' duration.

The small free pigmented bodies were very variable in numbers; they seemed more abundant in the chronic forms with cachexia.

Relation of those forms to the paroxysm.—Very many observations were made with a view of determining whether these organisms bore any definite relation to the remarkable periodic attacks which characterize acute malaria. For this purpose, in typical cases, examinations were made in the intervals of, just before, and in each stage of, the paroxysm. The results may be thus stated: there were instances, particularly if recent, in which the amœboid organisms were decidedly more numerous and larger before and during the paroxysms than in the intervals; there were others in which the number during the chill and hot stage was so small, that examples were very hard to find; in others again, slides taken before the attack and during each stage were negative, and yet in subsequent paroxysms the bodies were present in the blood. I think, on the whole, that pigmented bodies in the red corpuscles are more numerous before and during an attack, but the difference is by no means striking, and I have repeatedly had to search long in slides prepared during a paroxysm for a single example. In acute cases which have lasted some weeks, and have had no medicine, the amœboid bodies have seemed to be quite as abundant at one time as another. Nor have I been able to see any special difference in the form of the bodies just before or during the chill, though in the early days of the attack they may be small and less distinctly pigmented, or, indeed, may present, as in Case XXIX., already referred to, the appearance of vacuoles containing small solid bodies.

The remarkable segmentation of the rosette form was in each instance met with during the paroxysms, and Golgi claims to have traced in five cases a series of changes corresponding to the stages of the attacks. In the intervals, the pigmented bodies gradually increase in size until they fill the affected red corpuscles, and, finally, the pigment collects in the centre, as shown at Fig. 9. The process of fission coincides with the onset and course of the paroxysm, and by the time of its conclusion the rosette forms disappear. In Case LX.—a quartan ague—an attempt was

made to follow these changes, with the following result. The patient, a lad of 18, had had malaria, on and off, for a year, but for three weeks before admission the chills had been recurring with regularity. On Saturday, the 6th, the blood was examined in the chill. The red corpuscles contained many large pigmented bodies, and the rosette forms were numerous, many in process of subdivision. On the 7th and 8th, he was free from fever, and the most careful examinations of the blood failed to detect any forms but the ordinary pigmented intracellular bodies. They did not seem more numerous on the evening of the 8th than they were on the 7th. On the 9th, hourly examinations of the blood were made between 11 A. M., when the fever began, and 4 P. M. In the first two slides, there were very many pigmented bodies with the granules becoming concentrated, some with typical rosettes and a few in course of segmentation. In the specimens taken during the afternoon, the process of division was readily traced, and there were many of the small bodies in the field. On the 10th the note is: "No free bodies, all intracellular, tolerably numerous; no rosettes; no segmentation." On the 11th, several examinations were made, and the note reads, "absolutely none, save pigmented forms in the red." On the 12th, the slide at 8 A. M. showed large numbers of pigmented bodies, some with the granules irregularly arranged, other with distinct rosette. Fever began at 12 A. M. Throughout the paroxysm, hourly examinations were made; rosette forms were abundant, and segmentation active. On the 13th and 14th, the ordinary forms were present, and in the paroxysm of the 15th, the segmenting bodies were again seen. The development of pneumonia interrupted the observations. It is worth noting that in this case the onset of the paroxysms was marked by an outbreak of the most intense urticaria. Blood and lymph from the wheals did not show any special changes.

Certainly the segmentation seems associated in some way with the paroxysm in these cases, but unless our observations have been faulty or very incomplete, there are many others in which there are no such changes in the attack. It is a point, however, to which the attention of observers should be carefully directed.

The crescents appear, as already stated, to be confined to the more chronic cases, or to those which have had treatment. They may persist for weeks or months. Thus, in Case LVI. a patient had irregular fever with what he called dumb chills, which had lasted for a month; for three weeks there was fever without chills, the temperature rising on some occasions to 103°. The crescents were numerous, and were not associated with other forms. With this his general condition was good,

and he did not look anæmic. Under arsenic he improved, and the fever subsided, but the crescents were still in his blood six weeks from the date of the first observation.

Genuine paroxsyms may occur in these chronic cases without the development of other forms than the crescents. This observation was repeatedly made in Case XXV., a man with irregular malaria of many months' duration and occasional severe chills. The flagellate organisms did not seem to have any special relationship to the paroxysm, but they were so rarely seen that my observations on this point are not of much value.

Influence of medicines on the organisms.—Quinine invariably caused the pigmented bodies to disappear. In acute cases, which were usually studied during two or three paroxysms before the administration was begun, this observation was repeatedly confirmed. In a few days the corpuscles were entirely free; in several instances, the crescents appeared before the blood became normal. For example, Case XLVI. had his first chill on October 1st, and a daily recurrence until the 10th, when he came under observation. The pigmented bodies were abundant, and continued so on the 11th and 12th, when the temperature rose in the paroxysm to 105°. Quinine (twenty grains) was given on the morning of the 13th (which broke the chill), and repeated on succeeding days. The bodies were present on the 13th, and a few on the 14th. They were not found on subsequent days. In less acute cases the action of the quinine did not seem to be so prompt, and the crescents did not disappear so rapidly under its use. Certainly, in recent cases this medicine acts as a positive specific against these organisms, just as it does against the malady itself. Arsenic does not appear to influence the pigmented intracellular bodies. In a chronic case, without chills, but with irregular fever, the crescents persisted for over five weeks, although the patient had improved in general health and vigor, and was no longer anæmic. Thallin and antifebrin were given in some cases without any noticeable results. As is well known to practitioners in malarial regions, there are cases of intermittent fever which subside without special treatment. I have had several patients in whom, without any quinine, the chills stopped or recurred very irregularly. In Case LXVI., the crescents appeared in the blood, which at first contained only the intracellular forms.

Cases examined with negative result.—As before stated, there were eight instances of apparently true malaria in which the organisms were not found, and to these I shall now briefly refer. I would remark, in the first place, that we cannot always rely upon one, or even two, examina-

tions of the blood for these bodies. They may be very scanty, or they may be present at one examination and absent at the next. For example, Case XLI., a man, æt. 26, was admitted with a temperature of 104°. He had been cranberry-picking in New Jersey, and had been ill for a week with fever and indefinite pains, but no chills. He was so very dull, that as the fever persisted, typhoid was suspected, although, as a cranberry-picker, malaria was first thought of. The blood was examined on three occasions with negative results, but on the fourth observation, five days after admission, and when the temperature had fallen to normal, crescents were found, which continued in the blood until he was thoroughly cinchonized. The cases are as follows:

Case X.—Child, æt. 5; chills and fever in Maryland nine months ago, occasional chills since, the last two weeks ago; spleen four inches vertical diameter; had taken quinine, none recently. One examination.

Case XI.—Man, æt. 19; never malaria before. Four distinct paroxysms. Slides examined from fifth and sixth, taken in cold, hot, and sweating stages. No quinine. I did not see the case subsequently.

Case XX.—Man, æt. 40. First attack six months ago. Chill on and off for past three months. Blood examined three days after last chill. Had taken quinine for two days. Spleen enlarged.

Case XXI.—Man, æt. 28. Examined on 17th, first chill on September 6th; four since. On 14th, took quinine gr. xxx, and has had gr. x t. i. d. since.

Case XXVI.—Man, æt. 35. Chills for three weeks, at first quotidian, latterly tertian. Had taken medicine, but did not know the nature of it. Was admitted on 24th. Two examinations, negative; pigment in white corpuscles. On 25th quinine was given. Three subsequent examinations, without result.

Case XXVIII.—Man, æt. 60. Admitted on 14th. Well-marked chills for eight weeks; had one when he came in, and four after. Blood examined on 28th, two slides. He had had quinine gr. xx each day since admission.

Case XXXVIII.—Man, æt. 70, resident of the almshouse for six years. First chill on 2d, second on the 5th, third on 6th, when blood was examined, two slides.

Case LII.—Man, æt. 25. Chills and fever for six days. Blood examined in chill, and on the following day. Had had quinine.

Thus, in five of these cases quinine had been taken, and they may be counted out. In Case X., the child was brought from the country, and only one examination was made. Case XI. was undoubtedly a case of

quotidian ague, and the examination of slides taken from each stage of the fifth and sixth paroxysm was negative. I did not see the patient, and further examinations were not made. In Case XXXVIII., the bodies were not found on two occasions. This man also could not be followed, and I do not know his subsequent history.

The importance of excluding other causes for the paroxysmal chills was well illustrated by a case under the care of my colleague, Dr. J. H. Musser, which we regarded as one of malaria, but in which the pigmented bodies could not be found. The man had had chills on and off for several years; of late, the attacks had been more frequent and recurred more regularly. Quinine in medium-sized doses had no influence, but very large doses appeared to control the paroxysms. Their recurrence excited suspicion, and the discovery of pus in the urine, with decided pain on deep pressure in the lumbar region, indicated a more probable cause for the irregular chills.

Nature of the organisms.—It is very evident that we are dealing here with structures unlike any others which have been described in human blood, and with bodies which have no relation whatever to the spirilla, micrococci, and bacteria of certain acute diseases. I would call attention to the remarkable unanimity in the description of these parasites by Laveran, Richard, Marchiafava and Celli, Councilman, Golgi, and myself. Laveran's original description is well-nigh complete, and subsequent workers have done little else than confirm his results, though to Marchiafava and Celli is due the credit of insisting upon the amœboid character of the intracellular form. Before discussing the relation of the forms to each other, it will be necessary to take a brief review of cognate organisms occurring in the blood, upon which recent investigations throw an important light.

It has been known for some years that hæmatozoa exist in the frog; one form, a flagellate organism, the Trypanosoma sanguinis, described by Gruby in 1843, is a well-recognized monad; a second, the Drepanidium ranarum, of Lankester, is evidently a gregarine, possibly a larval form, as he suggests.[1] Having been long familiar with these bodies,[2] which were very abundant during several winters in the frogs in my laboratory at Montreal, I was at once struck with an apparent similarity to them of the forms found in malarial blood. The crescent-shaped body in particular resembles strongly certain of the gregarines, and I thought it possible that we had here an instance of a sporozoon becoming

[1] Quarterly Journal of Microscopical Science, vol. xxii.
[2] Canadian Naturalist, 1883.

flagellate at one stage of its development as Rivolta affirms may be the case. I soon discovered, however, that there were other observations on hæmatoza which bore more directly on the subject, and rendered possible a more likely explanation. Mitrophanow,[1] in 1883, announced the discovery, in the blood of the carp and of the mud-fish, of parasites belonging to the flagellate infusoria. A description of these forms need not detain us, further than to note that they were polymorphic, and one stage was represented by an amœboid body without flagella.

In a report published by the Punjab Government, December 3, 1880, and in the *Veterinary Journal*, London, 1881–82, my friend, Dr. Griffith Evans, described a new and very fatal disease known as *surra*, which prevailed among horses, mules, and camels in India, and in which he discovered a parasite in the blood during life. At first Evans believed it to be a spirillum, but subsequently came to the conclusion that it was a much higher organism. His observations have an important bearing on the question of the parasites in malaria. In 1885, Veterinary-Surgeon Steel published "An Investigation into an Obscure and Fatal Disease among Transport Mules in British Burma," which also proved to be *surra*. A careful clinical investigation of the disease led to the conclusion that it was a true relapsing fever, very similar to recurrent fever of man. Steel found the parasite described by Evans in all cases, and determined that it appeared as the temperature rose, and disappeared in the intervals between the paroxysms. He regarded it as a true spirillum, and named it Spirochæta Evansi. Both Steel and Evans found the disease readily communicable to dogs, horses, and mules, either by inoculation or by ingestion. Recently, on the return of Dr. Evans from India, he placed material from the *surra* disease in the hands of Dr. Crookshank, who has made an elaborate report,[2] confirming Dr. Evans's view that the organism is not a spirillum, and states that the parasite is morphologically identical with the hæmatoza described by Mitrophanow in the carp and mud-fish. In 1879, Lewis[3] described certain parasites in the blood of rats in India; and, again, in 1884,[4] he more fully discussed the question, and spoke of the identity of the organism with that found in the *surra* disease. Crookshank, in the paper just mentioned, gives the results of his investigations on the blood of European rats, twenty-five per cent. of which he finds infested with Lewis's parasite. It is a flagellate organism, with an undulating fin-like membrane, and is highly

[1] Biologisches Centralblatt, Bd. iii. p. 35
[2] Journal of the Royal Microscopical Society, 1886
[3] Quarterly Journal of Microscopical Science, 1879.
[4] Ibid, 1884

polymorphic. Crookshank has distinguished "globose, angular, non-filamentous, bi-flagellate, semicircular, and disk forms;" the latter represent the encysted stage. This organism is believed to be morphologically identical with the *surra* parasite and with Mitrophanow's hæmatozoa.

In the *Biologisches Centralblatt*, 1885, Professor Danielewsky, of Charkoff, makes an important contribution to the subject. He states that Trypanosoma, the well-known flagellate organism of frog's blood is polymorphic, and occurs in an amœboid form, and also produces spores; and, further, he has found in the red blood-corpuscles of birds a pigmented protoplasmic body, which subsequently appears in the plasma as a pigmented flagellate organism. In a later communication,[1] he suggests the identity of the pathogenic blood parasites of man with the hæmatozoa of healthy animals, and refers specially to the similarity of the forms which he has found in birds to certain of those described by Laveran in malaria.

With this information, we are in a better position to discuss the relation of the forms described to each other, and the zoölogical position of the organism. It is evidently closely allied to the hæmatozoa just spoken of, and the facts which we know of their life-history enable us to assert, with greater confidence, that we are here dealing with the varieties of a highly polymorphic species, and not with two or three different organisms. The flagellate form is doubtless the adult condition; and it is interesting to note, in contrast to the hæmatozoa of the rat and of the *surra* disease, the comparative infrequency of its occurrence. Laveran met with it ninety-two times in four hundred and thirty-two cases, and Councilman eleven times in eighty cases. The steps in development remain to be worked out. It seems clear, however, that the pigmented amœboid form may become transformed into a sporocyst (represented by the rosette form and its changes), or into an encysted body (resting form), the crescent. The gaps in our knowledge relate specially to the form and manner of entrance of the parasite into the red corpuscle. Do the solid particles contained in the vacuoles (Figs. 5 and 6) represent the earliest stage? I think it highly probable that they do, and that they, with the hyaline unpigmented bodies, are the immature forms. The spore-like structures which result from the segmentation of the rosette form do not resemble the small solid bodies seen in the red corpuscles, but are rather like the tiny free pigmented forms which, in some cases, were abundant in the plasma. Of the latter,

[1] Centralblatt f. die Medicinischen Wissenschaften, Nos. 41 and 42, 1886.

various sizes are found, and it is possible that from them the adult flagellate bodies arise. Golgi suggests that the spores, resulting from the segmentation pass to the spleen, and there attack the red corpuscles, in which they develop into amœboid forms. As at present the data are not available for a final decision, a further consideration of these points need not detain us. There is sufficient evidence to show that the various forms are only phases in the life-history of one, the flagellate protozoa, belonging to the order Flagellata-Pantostomata. Mitrophanow suggests a new genus, Hæmatomonas, to include the monad hæmatozoa; but Crookshank, who has carefully worked out the affinities of the parasites of the rat, the fish, and the *surra* disease, has referred them to the genus Trichomonas. The organism here described has not, however, the characteristic marks of a Trichomonas; for it lacks the undulating fringe on one side, and the caudal filament. Nor does it agree with the features of a Cercomonas; so that, meanwhile, until the true affinities are determined by an expert, its proper place seems to be the genus Hæmatomonas of Mitrophanow, which conveniently includes all monads parasitic in the blood. Thus: genus, Hæmatomonas; species, Hæmatomonas malariæ. Definition: Body plastic, ovoid, or globose, no differentiation of protoplasm, which contains pigment grains; flagella variable, from one to four. Highly polymorphic, occurring in (1) amœboid form; (2) crescents, encysted form; (3) sporocysts; (4) circular, free, pigmented bodies. The name designates the natural affinities of the parasite, the habitat, and the conditions under which it occurs, on which grounds it seems preferable to that of Plasmodium malariæ, suggested by Marchiafava and Celli.

Relation of the parasites to the disease.—The same difficulty meets us here as in so many affections in which microörganisms have been found: Are they pathogenic, or are they merely associated with the disease, which in some way furnishes conditions favorable to their growth? As evidence of their pathogenic nature may be urged, with Laveran, the constancy of their presence, their absence in other individuals in malarial regions, the destructive influence upon the blood-corpuscles, and their abundance in the graver forms of the disease. But even these considerations, weighty as they may appear, will not carry conviction to all, in the absence of experimental demonstration such as can be afforded in the case of certain pathogenic schizomycetes. Attempts to isolate and grow these hæmatozoa outside the body have failed. Marchiafava and Celli have shown that the inoculation of healthy persons with blood taken from a case of malaria is followed in a variable time by genuine ague paroxysms, in which the blood contains the parasites;

but in regions where malaria is prevalent such experiments are not wholly free from objections. A series of negative observations on undoubted cases of malaria would be convincing. I lay no special stress on the three cases in which I did not find the parasites, as the patients were not followed from day to day with the accuracy necessary to give any value to the observations. It must be borne in mind that hæmatozoa are not uncommon in animals, and, as in the rat, do not appear to interfere seriously with the health of their hosts. Under these circumstances, the association of a specific form with a definite disease in an animal makes it all the more probable that the species is pathogenic. A further study of the *surra* disease is particularly to be desired with the new light which Evans and Crookshank have thrown upon it. The conditions under which the disease occurs, combined with its paroxysmal character, are so similar to those of malaria, that a full explanation of its pathogeny would have a very direct bearing upon the present question.

To my mind, two facts in connection with these hæmatozoa point significantly to their etiological association with malaria. First, the positive anatomical changes which can be directly traced to their action, changes upon which one at least of the most marked symptoms of the disease depends. I refer to the destruction of the red blood-corpuscles, which can be followed in all its stages, and is as well defined an alteration of tissue brought about by a parasite, as any of which we know. The second fact is the action of quinine upon the parasites. The simultaneous disappearance of the symptoms of the disease and the hæmatozoa suggests that the specific influence of the medicine is upon the parasites, though it may be urged that the quinine, while curing the disease, simply removes the conditions which permit of their growth in the blood.

Practical considerations.—An interesting practical point is the diagnostic value of the presence of these bodies. There were six or eight cases in which the examination of the blood proved of great service in determining the existence of malaria. Some of these are worth mentioning. One of the first was a man æt. 37, who had been under observation on three or four occasions with anæmia and an enlarged spleen. He had had three attacks of hæmatemesis. There was no history of malaria, and, from the gravity of the case, I was led to regard it as one of severe splenic anæmia. On his fourth visit, however, a careful examination of the blood revealed the presence of the parasites, and I gave, in consequence, a more favorable prognosis in the case, which has since been justified. In an instance of pernicious malaria admitted to the Philadelphia Hospital, under the care of my colleague, Dr. J. H.

Musser, the diagnosis rested on the discovery in the blood of the characteristic changes in the corpuscles. To a third case, XL., I have already referred, and there were four or five other instances of chronic malaria in which the nature of the disease was determined by an examination of the blood. On the other hand, in many cases of suspected malaria, the absence of these bodies led to a more careful examination, and to the discovery of the cause of the chills and fever. Four of these were cases of phthisis with ill-defined physical signs; in a fifth, after several negative blood-examinations, the ague-like paroxysms were found to be due to a septic pneumonia; in a sixth and seventh, renal disease was discovered. I feel confident that, in malarial regions, the examination of the blood will prove, in skilled hands, a most valuable aid in the diagnosis of many obscure cases.

Melanæmia.—These researches on malaria throw light on the formation of pigment in the blood and various organs in the chronic cases. Evidently the primary change is in the red blood-corpuscle, which is gradually destroyed by the amœboid form of the parasite. Every stage of this process can be readily traced, and these observations bear out the more recent views on the origin of the pigment in the blood itself. The pigmentary degeneration of the red corpuscles noticed long ago by Frerichs and Kelsch,[1] was no doubt the same as here described. The gradual accumulation of the granules in the spleen, liver, and bone-marrow leads to the characteristic melanosis of these organs. I sought carefully for evidence of active interference with these parasites on the part of the white blood-corpuscles, but on only two or three occasions

Fig. 14.

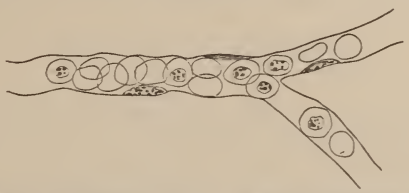
A colorless corpuscle studied for an hour and a half, during which time it had, as shown at 1, included a pigmented body, and was about to take another 2.

was this seen. Once a crescent was found inside a colorless corpuscle (Fig. 13), and again, as shown in Fig. 14, a corpuscle gradually enclosed two free pigmented bodies. The greater portion of the pigment resulting from the destruction of the monads is picked out by the cells of the spleen and bone-marrow, which also, no doubt, as in health, remove the effete red cells and their remnants. Pernicious malaria,

[1] Archiv d. Physiologie, 1875.

common enough when Stewardson[1] wrote his well-known article, has now become very rare in Philadelphia. In these cases, Marchiafava and Celli have found the capillaries of the various organs filled with corpuscles containing pigment-grains which appear enclosed in a hyaline matrix. Councilman and Abbot[2] have described the same change, and I am indebted to Dr. Councilman for the specimen from which the accompanying sketch was taken (Fig. 15). It represents a small brain-capillary filled with corpuscles, in many of which are pigmented bodies which stain deeply, and, so far as can be ascertained, are identical with

Fig. 15.



Sketch of a capillary vessel of gray matter of brain. Case of pernicious comatose malaria—Dr. Councilman. The red corpuscles are seen in outline, and in five there are pigmented bodies.

the pigmented organisms met with in the red corpuscles during life. Only one instance of fatal malaria came under observation, a man æt. 70, admitted to Dr. Musser's wards on October 25th. He had been on the Isthmus of Panama and in Georgia, and had chills and fever in both places; last chill was three days before admission. He had also had hæmaturia. He was very anæmic, the spleen was slightly enlarged, the temperature 101.3°. There was great stupor, and he was roused with difficulty; the tongue was dry. The temperature became subnormal on October 27th and 28th. Examination of the blood showed many pigmented bodies in the red corpuscles, numerous free circular forms, a few crescents, and several flagellate organisms. The stupor deepened to coma, and he died on the night of October 28th. The spleen and liver showed typical pigmentation, and the bone-marrow was also very dark. The spleen-pulp contained free pigment and many large cells, some of which were filled with dark granules, while in others there were bodies identical with the small pigmented forms so abundant in the blood during life. The marrow presented similar changes. The number of red corpuscles containing the pigmented bodies was not great, nor were the capillaries of the liver or the brain stuffed with them, as in the in-

[1] American Journal of the Medical Sciences, 1851.
[2] Ibid., 1885.

stances of pernicious malaria just referred to. Probably this was an instance of severe malarial cachexia of many months' duration, and scarcely should be grouped with the pernicious comatose form.

To my colleagues, Drs. Curtin, Neff, and Musser, I am indebted for the privilege of examining the malarial cases in their wards; and to my resident physicians, Drs. Donohue, Albertson, and Westcott, for assistance which materially lightened my work. *October* 28, 1886.

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9. *Cirrhosis of liver; fatal hemorrhage from œsophageal varix.*

Exhibited by Dr. W. Osler.

David M., æt. 44 years, white, admitted to Philadelphia Hospital Surgical Wards on October 26, 1886, with an ulcer of the leg. Had been a hard drinker, but up to the present time had not been unwell for many years. He had had syphilis. Patient was very pale and tremulous. On the morning of the 27th he complained of nausea, and at 1 o'clock P.M. vomited a large quantity of blood. The vomiting continued at intervals through the afternoon and night, and he lost several pounds of blood. I saw him for the first time at noon on the 28th, when he was in a semicomatose state, could not be moved, was breathing deeply, and was evidently failing fast. The examination of the thoracic viscera was negative. In the abdomen there was notable diminution in the area of liver dulness, and it was considered probable that this was the cause of the hemorrhage. The blood, which he had last vomited, had been kept, and was thickly clotted, dark in color, and not mixed with food; that first brought up was brighter, and contained part of his dinner. A stool, which he passed to-day, was dark and tar-like. He gradually sank, and died on the 29th, with no further bleeding.

The autopsy showed advanced cirrhosis of the liver of the ordinary atrophic form; the portal branches within the organ were very much contracted. The diaphragmatic veins were greatly distended and anastomosed freely with the œsophageal plexus. A rich network of vessels covered the kidneys, and the posterior and lateral folds of the peritoneum, particularly in the region of the descending colon. The stomach did not contain blood; mucosa was pale; no erosions. The veins about the cardiac end were greatly distended. The œsophagus presented in its lower half a network of dilated veins, some of which projected on the mucosa. One of these, the size of a quill, extended in a tortuous

manner along the posterior wall, and at its upper part showed a grayish elevated spot, covered with a thrombus. This proved to be an erosion on the vein, and a probe passed freely from the veins through the orifice. This, doubtless, was the cause of the hemorrhage.

Remarks.—Bleeding from an œsophageal pile is a rare, but well recognized event in cirrhosis of the liver. It is by no means uncommon to find the veins of the gullet greatly distended in this disease, as their anastomoses with the gastric and the azygos vessels affords one important channel by which the portal blood reaches the general circulation. In cases of fatal hæmatemesis a careful inspection of the œsophagus should always be made, otherwise the cause of the bleeding may be overlooked. *February* 24, 1887.

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10. *Notes on case of twins united by abdominal sac.*

Exhibited by Dr. J. Collins.

February 16, 1887, I was called to see Mrs. S., who had engaged me for attendance in the latter part of February. On my first visit she informed me that she had had pains all day, also thought that she had been losing water from the uterine cavity. At the time, 7 P.M., the uterus was only partially dilated. No point presenting recognizable, there was nothing to do but wait.

About 11 P.M. pains became severe; failing to recognize any kind of a presentation that declared for either end of the fœtal body, I determined to deliver as no signs of life could be detected. Ether was given to relaxation. On introducing my left hand into the uterus I discovered a hand and foot. On each pain a sac would bulge into the uterine orifice. I was soon convinced that the hand and foot belonged to separate individuals, but by carefully following the limb of the lower extremity, and changing hands during the procedure, I found the feet that belonged to the same individual. At this point a strong pain, a little traction and rotation.

Delivered the first one, dead, with open abdomen, liver, kidney, etc., expressed without abdominal covering. There was no cord connected with this child.

A few moments after, the second child was delivered by rapid procedure, also dead. From the usual point there was no umbilical cord. There was but one cord, which seemed to be attached near the apparent vulva of this child.

The mother requiring some attention after the placenta was delivered, in her haste to dispose of things promptly, the nurse placed the placenta beyond my reach, which I have sincerely regretted.

The father and mother are German; the mother has given birth to two healthy children; had one miscarriage since her son was born, who is now about two years old. The father is in good health, vigorous, and strong; habits good. Why this turning aside from the usual course of events into the domain of teratogeny is a wonder.

Anatomical description by Dr. H. F. Formad. Dr. Collins's specimens of "Twins united by an Abdominal Sac," which I dissected with his kind permission, and preserved in good state for future studies, belongs to the same class of twin-monstrosities as the Siamese Twins, viz., the *xipho-pagi*. These are characterized by two developed individuals united anteriorly only by their soft parts, sometimes only by the integuments or by fibrous bands.

In Dr. Collins's case the individuals being united anteriorly by means of the genito-urinary viscera, as well as by the abdomen, belongs to that form of xipho-pagus which is termed *omphalo-ischio-pagus*. There was only one placenta and one umbilical cord (which is the rule in this form of double monstrosities). The twins here were both females at full term, or nearly so, and must have been alive up to a few moments before delivery. Size of children somewhat below normal; one being quite larger than its fellow. The larger one was more perfect in bodily development and figure, save in its lower part of abdomen and genitals. The smaller child had its body distorted, figure of S-like, due to the cramped and twisted position it occupied in utero around the trunk of its fellow. It had a right anterior curvature of the lumbar region of the spinal column to such an extent as to bring it forward to a plane with the anterior portion of the pelvis. The ribs on the right side were strongly flexed upon themselves, so as to leave hardly any space for a pleural cavity on that side. The bones of the extremities were also more or less curved in this child. As in the larger child, the pelvis was deficient anteriorly, there being no symphysis.

The bond of union between the two infants was a membranous sac or cone-shaped soft tube that merged into or with the abdominal integuments of the larger child by its narrower end, just above the pubis, and with those of the smaller, crippled child, by its wider end, so that the whole abdominal front wall of this latter child was substituted and covered by the connecting sac. The tissue of the sac was nearly transparent, and its inner lining was continuous and communicating with the peritoneal cavities of both children. Length of connecting sac

about six inches (it had been severed during the delivery of the children). The abdominal viscera were protruding outside of the body in the smaller child, but it was evident that they were contained in the connecting abdominal sac, as there was no room for them in the crippled small peritoneal cavity. The abdominal viscera belonging to the larger child were in their natural place.

There were no external or natural orifices for either the genito-urinary or alimentary canal in either of the twins, though each had its own more or less complete internal viscera. The rectum and the urethra of

Fig. 1.

each child terminated or opened directly into the connecting membranous sac between them. The uterus of each was bicornate, and in both children lay outside the body within the membranous sac, and having one common vagina that formed a bridge between the two individuals. The umbilical cord entered also through the sac.

Outside of these anomalous conditions the larger child had normal organs, as regards position and structure.

The smaller child had only the thoracic organs and head in more or

less normal position and state, while the abdominal organs, as stated before, were situated in the membranous sac, save the kidneys and suprarenal bodies. All the organs of the body, including the thoracic,

Fig. 2.

were more or less malformed, and part of the diaphragm was absent.

Closer studies could not be made, as the specimens, particularly the connecting sac, were somewhat damaged during the delivery of the infants. *March* 10, 1887.

11. *The structure of white thrombi.*

Read by Dr. W. H. Welch,

A year ago, upon an occasion similar to this, you had the pleasure of listening to Dr. J. Collins Warren's address upon the healing of arteries after ligature. As his researches were directed especially toward the later stages of the changes which follow injury of the blood-vessels, it will, perhaps, not be unacceptable if I call your attention to the histological structure of those plugs which often constitute the earliest alteration following such injury.

While all that pertains to the subject of thrombosis is of importance, recent investigations have lent especial interest to the study of the minute structure and the mode of formation of white thrombi.

Since Virchow's memorable publications[1] upon the subject of thrombosis, it has been generally believed that a thrombus is essentially a blood coagulum, and differs from an ordinary post-mortem clot only in the arrangement and the relative proportion of the constituent histological elements. The most important of the differences noted by Virchow are the characteristic lamination of thrombi, and their greater richness in white blood-corpuscles, and in granular material. These differences were believed to be sufficiently explained by the slow formation of thrombi from the circulating blood, in contrast with the rapid coagulation of blood at rest, and by secondary changes in the thrombus.

During the two decades following the publication of Virchow's researches on this subject, more attention was paid to the causes, to the effects, and to the metamorphoses of thrombi, more particularly to their so-called organization, than to the intimate structure of recently formed thrombi. Zahn's investigations of thrombosis, published in 1875, marked an epoch in the history of our subject.[2] Zahn had been preceded by Mantegazza,[3] who, in 1869, called attention to the rôle played by white blood-corpuscles in the formation of white thrombi, but the observations of the latter author had remained comparatively unknown.

Zahn emphasized the most important differences existing between thrombi formed from the blood in repose, the so-called red thrombi, and those developed from circulating blood, viz., the white and the mixed

[1] Virchow: Gesammelte Abhandlungen. Frankfurt a. M., 1856.
[2] Zahn: Virchow's Archiv, 1875, Bd. 62, p. 81.
[3] Mantegazza: Gaz. med. Lombarda, 1869.

thrombi. Whereas the former do not differ from an ordinary coagulum of blood, the latter, according to Zahn, originate from clumps of white corpuscles. Zahn observed microscopically in the mesenteric vessels of the living frog, the first formation of white thrombi out of white blood-corpuscles which accumulated in vessels at places which had been subjected to various injuries. The white corpuscles thus accumulated, if they were not detached by the circulation, rapidly disintegrated into a mass of granular material which Zahn considered to be granular fibrin. According to the widely accepted views of Zahn, therefore, a white thrombus at its inception consists essentially of white blood-corpuscles, which, after a short time, break up into a mass of granules identical with fibrin in their reactions.

The observations of Mantegazza and of Zahn were confirmed, in 1876, by Pitres,[1] who made corresponding observations of the living circulation in warm-blooded animals, whereas Zahn studied the circulation only in frogs. Pitres, however, did not, like Zahn, identify the granular material resulting from the disintegration of white blood-corpuscles with fibrin.

The rôle thus assigned to the white corpuscles in the formation of white thrombi certainly seemed to be at variance with Virchow's view that all thrombi are coagula. A reconciliation, however, was effected between the new observations and the old doctrine, chiefly through the investigations of Weigert.[2] This pathologist, adopting the views of A. Schmidt as to the part taken by white corpuscles in the spontaneous coagulation of the blood, assigned to these corpuscles essentially the same rôle in white thrombi. The coagulation necrosis of leucocytes in thrombi is a process differing, according to Weigert, morphologically, but not in essence, from the dissolution of white corpuscles and the formation of fibrillated fibrin in the ordinary coagulation of the blood. White thrombi, therefore, continued to be regarded as in the main genuine coagula.

The first opposition to the views of Zahn came from Hayem,[3] who, in 1878, attempted to prove that the coagulation of fibrin is a function of the small bodies, called by him hæmatoblasts, and subsequently, by Bizzozero, blood plates, the name now generally adopted. Osler, who was among the first to observe the existence of human thrombi composed almost exclusively of blood plates (or plâques, as he, accepting the

[1] Pitres: Arch. de Phys. norm. et path., 1876, p. 230.

[2] Weigert: Virchow's Archiv, Bd. 70, 1877, and Bd. 79, 1880 Fortschritte d. Medicin, 1883.

[3] Hayem: Recherches sur l'Anatomie norm. et path. du Sang. Paris, 1878. Comptes Rendus de l'Acad. d. Sc., 1882, 18 Juli.

suggestion of Kemp, prefers to call them), has presented fully in the last series of Cartwright Lectures, the existing state of our knowledge concerning these bodies.[1] In 1882, Hayem published his observations on the structure of thrombi. He found that the thrombi which are formed in wounds of arteries are made up of blood plates.

A few months later Bizzozero[2] described, with much detail, both the fibrin-forming properties of the blood plates, and their presence as the essential and primary constituent of white thrombi, in these respects confirming the opinions of Hayem. Bizzozero was the first to study the formation of thrombi from blood plates in the living circulation, using for this purpose the mesentery of warm-blooded animals.

In the following year, Hlava,[3] working under Weigert's direction, was unable to confirm the views of Hayem, and of Bizzozero, and upheld the doctrine of Zahn and of Weigert, that white thrombi, in their earliest formation, consist mainly of leucocytes, which subsequently undergo coagulation necrosis.

Lubnitzky,[4] working under the direction of Langhans, published, in 1885, an interesting article, in which she claimed that the thrombi which are formed in arterial wounds, and which are the chief agent of nature in checking hemorrhage from this source, are composed primarily of blood-plates. The blood-plates, when thus accumulated, are, according to Lubnitzky, either identical with fibrin, or are quickly transformed into this substance.

The most thorough study hitherto made of the share taken by blood-plates in the formation of thrombi we owe to Eberth and Schimmelbusch.[5] These authors consider that sufficient proof of the existence of blood-plates in the normal circulation is afforded by the observation of the plates in the circulating blood of the mesenteric vessels of dogs and rabbits examined under physiological salt solution, with high magnifying powers. In opposition to Hayem and to Bizzozero, they deny that the plates have any share in the coagulation of fibrin, which they regard rather as a kind of crystallization in the plasma. The plates, when removed from the natural conditions of their existence, rapidly undergo a metamorphosis, called by Eberth and Schimmelbusch viscous metamorphosis, and characterized especially by the sticking of the plates to each other and to foreign substances. Under normal conditions the

[1] Osler: On Certain Problems in the Physiology of the Blood Corpuscles. The Medical News, April 3, 10, 17, 1886.

[2] Bizzozero: Virchow's Archiv, 1882, Bd. 90, p 261.

[3] Hlava: Arch. f. exp. Path. u. Pharm., 1883, Bd. 17, p. 392.

[4] Lubnitzky: Arch. f. exp. Path. u. Pharm., 1885, Bd. 19, p. 185.

[5] Eberth u. Schimmelbusch: Virchow's Archiv, 1885, Bd. 101; 1886, Bd. 103, Bd. 105.

plates circulate with the red corpuscles in the axial blood current, but they make their appearance in the plasmatic zone when the rapidity of the circulation is diminished. A moderate slowing of the blood current is attended by the formation of the so-called border zones, or accumulation of white corpuscles in the plasmatic current, whereas a greater diminution of the velocity of the stream is characterized by fewer leucocytes, and more plates in the peripheral current. Other irregularities of the circulation, such as the little eddies produced by obstacles or projections in the stream, or by dilatations of its bed, may likewise throw the plates from the axis into the periphery of the stream. Mere slowing of the circulation is not attended by the formation of thrombi. In order to observe this formation, Eberth and Schimmelbusch subjected the living mesenteric vessels, chiefly of dogs, to various mechanical and chemical injuries. They then observed under the microscope, in many, but not in all instances, the accumulation of blood plates at the seat of injury. Here the plates became adherent to each other and to the wall of the vessel, in consequence of their viscous metamorphosis, and thus formed plugs which were often subsequently washed away, but which sometimes increased in size so as to obstruct completely the lumen of the vessel. Red and white corpuscles may be included in the mass of plates, but their presence is purely accidental, and they are not to be regarded as an essential constituent of the primary thrombus.

As the result of their microscopical observations of the formation of thrombi in living bloodvessels of warm-blooded animals, Eberth and Schimmelbusch, therefore, conclude that white thrombi are at first composed essentially of blood plates, and that the chief factors in the causation of such thrombi are slowing of the circulation or other irregularities in the current, and the viscous metamorphosis of the blood plates. This metamorphosis may be the result of various influences, such as contact with injured or diseased vascular walls and with foreign substances.

These conclusions as to the structure of white thrombi at their earliest formation Eberth and Schimmelbusch confirmed by the microscopical examination of sections of thrombi produced artificially by various injuries to the vessels. In experimental thrombi produced by mechanical injury of the vessels, as by wounds or by temporary ligation, they failed to find any fibrillated fibrin, whereas, in thrombi formed around foreign bodies introduced into the lumen of a bloodvessel, they observed some fibrin, situated usually between masses of plates, although even here they think it probable that fibrin is absent in the very earliest stages. They also detected fibrin, but in less amount, in thrombi produced by cauterization of the vessel walls.

While the investigations of Eberth and Schimmelbusch confirm the view of Hayem, Bizzozero, and Lubnitzky that white thrombi are made up primarily of an accumulation of blood plates and not of leucocytes as Zahn had led us to believe,[1] they are opposed in one important particular to the conclusions of the latter group of authors. They deny that the blood plates are in any way concerned in the generation of fibrin or are transformed into a substance resembling fibrin. They, therefore, deny that a white thrombus is primarily a coagulum, as has hitherto been unquestionably believed. They regard the process of thrombosis, here under consideration, as a *conglutination* of bodies pre-existent in the blood and not as a coagulation.

The arguments brought forward by the preceding investigators in favor of the existence of blood plates in large number in the normal circulation, convincing as they may seem, are nevertheless opposed by several observers. In view of the researches of Löwit,[2] this must for the present be considered as an open question.

Notwithstanding the brief period which has elapsed since the publication of Eberth and Schimmelbusch's researches upon thrombosis, their conclusions have already met with considerable opposition. It was hardly to be expected that such a radical overturning of accepted beliefs as these recent investigations involve should pass unchallenged.

While there is general agreement of opinion as to the important participation of blood plates in the composition of white thrombi, Eberth and Schimmelbusch's conception of the process of thrombosis as a conglutination of blood plates which have undergone a viscous metamorphosis is opposed by Hanau[3] on the ground that thrombi never have a viscid consistence. In support of the coagulative nature of the accumulation and metamorphoses of blood plates in white thrombi Hanau finds that plates as well as fibrin are transformed into hyaline, that a rim of hyaline forms around masses of plates, and that fibrin and plates often take the place one of the other in thrombi.

Weigert[4] protests even more vigorously against the effort of Eberth

[1] Since the delivery of this address Eberth and Schimmelbusch have published the results of their repetition of Zahn's experiments on the mesenteric vessels of frogs, and they find that fusiform corpuscles, which they consider to correspond to the mammalian blood plates, are the main constituents of white thrombi artificially produced in these animals. Vide Virchow's Archiv, Bd. 108, 1887. Löwit, on the other hand, regards these fusiform corpuscles as a variety of the white corpuscles and not as the analogues of blood plates, and he confirms the original statements of Zahn regarding the formation of white thrombi in frogs. Archiv f. exp. Path. u. Pharm. Bd. 23, 1887.

[2] Löwit: Beiträge z. Lehre von d. Blutgerinnung, Sitzb. d. k Akad d. Wiss. Wien, Bd. 89, Abth. iii. u. Bd. 90, Abth. iii, and Tageblatt d. 59ter Versaml Deutscher Naturforscher u. Aertze in Berlin, p 306, 1886.

[3] Hanau: Fortschritte der Medicin. No. 3, 1887

[4] Weigert: Tageblatt der 59ter Versamml. Deutscher Naturforscher u Aerzte in Berlin, p. 306, 1886.

and Schimmelbusch to remove a large class of thrombi from the category of coagula. He has made a careful examination of human white thrombi, and points out especially their richness in fibrillated fibrin, which he demonstrates by a new staining process, and the abundance of leucocytes. He is unable to identify these anatomical thrombi with the experimental thrombi of Eberth and Schimmelbusch, and argues that until some reconciliation is effected between the two we should continue to base our conception of the nature of thrombi upon the study of the anatomical thrombi. Eberth and Schimmelbusch reply that their studies have been directed to the very earliest stages of the process of thrombosis, whereas the thrombi examined by Weigert belonged to subsequent metamorphoses.[1]

It is apparent from the foregoing review of recent investigations concerning the nature and structure of thrombi that unanimity of opinion on this subject has not been reached. There is general agreement that the blood plates play an important rôle in the early formation of many thrombi. Further investigations are needed to determine whether or not the plates are present in the perfectly normal circulation. For a proper understanding of the process of thrombosis it is important to determine whether or not the blood plates when accumulated to form a thrombus, are products of coagulation or subsequently undergo any metamorphosis which can be called coagulation. To determine this the gross characters of the plate thrombi, such as their color and consistence, will serve as important criteria, as has been pointed out by Weigert. It is, of course, of capital importance to learn whether the experimental white thrombi differ in their nature from human thrombi as seems to be intimated by Weigert. Before far-reaching conclusions can be drawn it is necessary to demonstrate the identity of the experimental and of the anatomical process of thrombosis. The microscopical study of human thrombi certainly seems opposed to the opinion that fibrin and leucocytes are unessential constituents of white thrombi. So constant and so abundant are these elements in post-mortem white thrombi that pathological anatomists will not readily admit that their presence is accidental or unessential to our conception of the nature of the thrombotic process.

In view of the fundamental importance of the question last touched upon, I have undertaken some investigations, first, as to the structure of

[1] Schimmelbusch: Tageblatt d. 59ter Versamml Deutscher Naturforscher in Berlin, p. 306, 1886. Eberth u. Schimmelbusch, Fortschritte der Medicin, No. 6, 1887. The paper of Löwit, on thrombosis, who is opposed in many important particulars to Eberth and Schimmelbusch, appeared after the delivery of this address. Arch f. exp. Path. u. Pharmak. Bd. 22, 1887.

human white thrombi; and second, as to the structure of thrombi produced experimentally in animals by mechanical injury of the blood-vessels. In the study of experimental thrombi I have directed my attention, in the first place, to their constitution at their earliest formation, and especially to the presence or absence of fibrin and of leucocytes at this period. It has seemed to me that a control with reference to the latter point of the observations of Hayem, Bizzozero, Lubnitzky, and especially of Eberth and Schimmelbusch, notwithstanding the carefulness of these observations, might not be unwelcome. I have also studied the structure of experimental thrombi in their later stages. It is undoubtedly upon this point that our knowledge is the least complete, and it is to be expected that when this gap is filled up there will be less divergence of opinion as to the relation between the experimental and the human thrombi.

There will be found on exhibition under the microscopes sections of human marantic thrombi formed in various infectious and wasting diseases. Among others specimens are present from a case of widespread thrombosis following parturition. In this case there were fresh thrombi in the femoral and iliac veins, the inferior vena cava, the branches of the pulmonary artery, and the cerebral sinuses. The constituent elements of these thrombi are fibrillated fibrin, hyaline substance, red blood-corpuscles, leucocytes, fragmented nuclei, and granular material, of which a considerable part can be recognized as blood plates. The proportion of each of these elements in the composition of thrombi varies much in different cases, and it will be well to consider the share taken by each in the formation of thrombi.

There have been various opinions as to the nature of the granular material found in thrombi. Thus it has been regarded as produced by the breaking up of fibrillated fibrin (Virchow), as molecular or granular fibrin deposited as such from the blood—a view advocated by the majority of the older (Mandl, Addison) and by many recent authors—as granular fibrin formed by the necrosis of white corpuscles (Zahn), as the result of simple disintegration of white corpuscles (Pitres). At present, however, there can be no doubt that most of what has been called in thrombi granular fibrin, or the products of disintegration of leucocytes, consists of more or less altered blood plates. The acquisition of this knowledge is an important advance in pathology, whatever may be thought of the nature of the plates.

Blood plates seem to be a constant constituent of fresh marantic thrombi. The plates are often present in such thrombi in as recognizable form and arrangement as in recent experimental thrombi. I have

found thrombi, particularly some endocardial vegetations and parietal arterial thrombi, which at first glance appear to be composed of nothing but plates; but careful examination in such cases has always revealed the presence also of fibrillated fibrin and leucocytes. In the majority of cases, however, the part of the thrombus composed of plates is less extensive then that made up of fibrin and leucocytes. Frequently the plates are arranged in masses between which lie the fibrin and leucocytes. Such masses of plates, which are more frequently situated in the interior of the thrombus than adjacent to the vessel-wall, are often enveloped in a rim of dense material resembling fibrin. In sections stained with hæmatoxylin and eosin the areas occupied by the plates can often be recognized with a low power by the various manner in which the different constituents of the thrombus stain.

All of the granular material in thrombi cannot be demonstrated to be composed of plates, but it is probable that most of this formless granular matter is the result of the disintegration and metamorphosis of the plates. That some of the granules are produced by the disintegration of leucocytes is probable, for it is not difficult to demonstrate the destruction of leucocytes in many thrombi. I believe also that a granular precipitate in thrombi is sometimes caused by the hardening agents.

As regards fibrin, I can confirm the recent statements of Weigert as to the abundance and the constant presence of this substance in all marantic thrombi, except in softened foci where it is absent. Some thrombi are composed almost wholly of fibrin. The fibrin may assume various forms, such as the form of a delicate network, or of coarse interlacing or parallel bands, or of irregular masses, or of the so-called canalized fibrin. In sufficiently thin sections, such as can be made from specimens imbedded in paraffine, there is generally no difficulty in demonstrating in thrombi a rich network of fibrin even without the aid of Weigert's special stain for this purpose. Leaving out of question, therefore, the nature of the blood-plates there can be no doubt that human thrombi, as we meet them at autopsies, are genuine coagula, save in the foci of so-called puriform softening.

Hyaline material appears to be formed both out of fibrin and out of blood plates. Thrombi composed wholly of hyaline I have found in the liver of a cat in which a few drops of croton-oil had been injected, in hemorrhagic infarctions of the lungs, and in corroding ulcers of the duodenum and of the stomach. Hyaline is an inconstant constituent of thrombi, but its presence is not rare.

The accumulation of leucocytes in human white thrombi is so well known that there would be no necessity of emphasizing it here, were it

not that the recent study of experimental thrombi has led to a revision of the doctrine that white thrombi are composed primarily of masses of white blood-corpuscles. While it is true, as has already been mentioned, that there are thrombi which are composed almost entirely of plates, or of fibrin, or of hyaline, or of these substances in combination, this is the exception, and in the vast majority of fresh thrombi leucocytes are present in large number. In inflammatory thrombi leucocytes may be so abundant as to obscure all other constituents. Usually the leucocytes in marantic thrombi are not scattered uniformly throughout the thrombus, but are massed together in clumps; these clumps of leucocytes, unlike the clumps of plates, are generally pervaded by a network of fibrin.

It is not at all infrequent to find in old thrombi leucocytes and even masses of them which are devoid of nuclei. In undergoing this necrosis the nuclei of the white blood-corpuscles may be broken up into fragments which can be recognized as small irregular particles which assume a nuclear staining, but this nuclear fragmentation does not seem to be the rule. Generally the necrotic leucocytes can be recognized simply by their form, without any trace of nuclei.

Red corpuscles are present in variable numbers in marantic thrombi. They cannot be regarded as an essential constituent of the thrombus. I find in many marantic thrombi the so-called shadows of the red blood-corpuscles, which can be easily overlooked unless especial attention be given to searching for them.

In properly prepared sections it is not rare to find colonies of micrococci even in thrombi not connected with pyæmic processes, especially in marantic thrombi from cases of typhoid fever or other infectious diseases.

From the foregoing summary of the histological characters of human white thrombi, it is apparent that any satisfactory explanation of the process of thrombosis must account for the presence of blood plates, of fibrin, and of leucocytes, for these are essential constituents of thrombi. The valuable investigations by Eberth and Schimmelbusch of experimental thrombi have led them to regard the blood plates as the sole primary elements in these plugs. Further investigations are needed to determine the rôle played by fibrin and white blood-corpuscles in the formation of experimental thrombi.

My experiments upon the production of thrombi have been made mostly on dogs. The vessels selected have been the femoral artery, the femoral vein, and the jugular vein, in the majority of cases the femoral vessels. Various methods were employed to produce thrombi, such as

the application of caustics, the introduction of foreign bodies, and various mechanical injuries. I have given the most attention to the thrombi resulting from mechanical injuries, for it is admitted by Eberth and Schimmelbusch that the thrombi following the application of caustics, and those formed around foreign substances, contain, in an early stage, if not at their beginning, fibrin as well as blood plates. These authors assert that "coagulation is a process which plays only a modest rôle in the circulating blood, whereas here the most prominent and frequent phenomenon is conglutination, which, indeed, is solely concerned in the practically most important form of thrombosis, viz., that following simple mechanical injury of the vessel-wall, in whatever way this may have been produced."[1]

The following two modes of producing mechanically thrombi have given good results. One method is to tie a stout ligature tightly and suddenly around the vessel and at once cut the ligature loose. In this way the intima and a part of the media are usually torn. It is only when great force is used that the vessel is ruptured. The ligature leaves a whitish ring around the vessel at the seat of its application. If, as often happens, the walls of the vessel remain stuck together after removal of the ligature, then moulding the vessel slightly between the fingers will restore the lumen, which now presents a fusiform dilatation at the seat of injury. The adventitia in this situation often becomes infiltrated with blood.

Another method which I have employed, is to push into a branch of the femoral artery or femoral vein one of the hooked instruments or gouges employed by dentists and called excavators, and then, after pressing the hooked extremity forward into the main trunk to scrape the inner wall of the vessel to any extent desired. The stem of the instrument, if necessary, can be filed down so as to render its introduction easier. After the withdrawal of the instrument the opened branch of the vessel is secured by two ligatures. Of the various shapes which the working extremities of these instruments possess, those with a small cup-shaped gouge bent at right angles to the handle (spoon excavator) I have found particularly suitable.

The animals experimented upon have been tied down and anæsthetized, usually with morphine and chloral, or morphine and ether, or morphine alone.

It is important, when the vessel is removed from the body, that four ligatures should be applied, two above and two below the seat of injury,

[1] Eberth and Schimmelbusch. Virchow's Archiv, Bd. 105, p. 459.

and that any collateral branches included between the pairs of ligatures should also be tied. These ligatures should be applied with as little disturbance of the vessel as possible. If the vessel be cut out without the preliminary application of ligatures, the contents of the vessel are partly discharged, and in this way the thrombus may be lost or its position changed.

Various hardening fluids were employed, such as corrosive sublimate, alcohol, Müller's fluid, picric acid, osmic acid, and Flemming's solution. Of these, warm saturated solutions of corrosive sublimate are decidedly the best. This fluid preserves the blood plates and other elements almost perfectly and admits satisfactory subsequent staining of the specimens.

The procedure adopted in hardening in corrosive sublimate is the following: A clear, saturated aqueous solution containing some undissolved sublimate at the bottom is heated to 40° C., and in this is suspended the specimen to be hardened. After a few minutes I have usually cut away the ligatures at the ends of the vessel, as there is now no danger of the escape of the contents. The vessel containing the sublimate solution and specimen is kept in a thermostat at a temperature of 40° for one to two hours. The specimen is then washed in water having a temperature of 40° and afterward placed in a mixture of half alcohol and half water, and kept in this mixture at a temperature of 40° for a number of hours, often over night. This prolonged washing is to remove crystals which otherwise are present in large number. Even after this treatment sometimes peculiar crystals are present, which, however, do not materially interfere with the study of the sections. The addition of a little iodine to the washing fluid, as suggested to me by Prof. Gaule, assists in removing the crystals. From the fifty per cent. alcohol the specimen is transferred to strong, and finally to absolute alcohol. I have made use almost exclusively of paraffine as an imbedding medium, as much thinner sections can be obtained in this way than by imbedding in celloidine. Satisfactory results can be obtained by staining the specimens *en masse* in hæmatoxylin and in eosin, but, as a rule, the sections have been stained after causing them to adhere to the slide. When serial sections were desired, they were cut in the form of ribbons. Gaule's method of making the sections adhere to the slide by placing upon each section laid dry upon the slide a drop or two of forty or fifty per cent. alcohol, and after ten minutes putting the slides in a thermostat at a temperature of 40° and keeping them there for two hours, is the simplest and best with which I am acquainted. After this treatment the sections are so firmly adherent that all the manipulations of staining and preparing the sections for mounting can be carried on without fear of their detachment.

In sections stained with hæmatoxylin and eosin the plates have a violet tint, and when in masses can be readily recognized with a low power.

I wish first to direct your attention to the macroscopic and the microscopic appearances of fresh experimental plate thrombi. Such a thrombus may be conveniently produced by tying forcibly a coarse string around the femoral artery of a dog and then at once cutting the string loose in the manner already described. After the time desired for the production of the thrombus has elapsed, the injured part of the artery inclosed between two pairs of ligatures is to be removed and the artery is to be carefully slit up with a pair of delicate scissors.

Let us examine an artery treated in this way which contains a thrombus formed within five minutes. There will be found, closely adherent to the torn inner wall of the vessel, a parietal thrombus which at this period does not usually extend in a longitudinal direction much beyond the ring of lacerated tissue. The thrombus can be readily distinguished by its color from the blood which envelops it and which can be washed away with salt solution. The thrombus projects irregularly into the lumen of the vessel, the projecting part being made up usually of round or irregular masses which are connected together.

The thrombus has a homogeneous, grayish, translucent appearance, comparable to particles of boiled sago, and resembling, therefore, the color of the Malpighian bodies in a waxy spleen. When bits of the thrombus are pressed into thin layers between the slide and the cover-glass they present a bluish transparency almost glass-like. The epithet hyaline can be appropriately applied to the naked-eye appearance of the thrombus.

The consistence of the thrombus is soft, the weight of the cover-glass sufficing to make bits of the thrombus spread out into thin layers. In attempting to tease apart portions of the thrombus, it is found that this does not break up into little granules, as would be the case if the blood-plates which compose it had undergone no changes and were simply in apposition to each other; but, on the other hand, the thrombus possesses considerable cohesion, and in breaking it into fragments with teasing needles fine sticky threads can be drawn out a short distance which break apart, or, if the tension be removed, retract. Portions of the thrombus pressed between the fingers present a sticky, gelatinous consistence. In view of Hanau's objection, already mentioned, to Eberth and Schimmelbusch's designation of the change by which the plates adhere to each other as a viscous metamorphosis, it is to be emphasized that fresh plate thrombi have a somewhat viscid consistence, which becomes more marked in the course of an hour after the removal of the

thrombus. I cannot, therefore, accept Hanau's objection, so far as this point is concerned, as valid.

If parts of the fresh thrombus be teased in physiological salt solution or in Bizzozero's methyl-violet salt solution, or in Hayem's solution, there will be seen masses of blood plates and a large number of free plates floating in the liquid. The plates appear as pale, well differentiated, round or somewhat irregular bodies varying in size, the average being about one-quarter the diameter of a red blood-corpuscle. Masses of plates resemble colonies of large micrococci. They can be made to assume feeble tints with a variety of coloring agents, but I have not been able to give them, in their fresh state, a sharp, elective staining. In water the plates become paler and somewhat swollen; in very dilute acetic acid they become darker in color and more distinct, in strong acetic acid they disappear from view.

By tapping with a needle upon the cover-glass it can be seen that the individuals composing the masses of plates adhere to each other. Such masses may be readily flattened out and compressed. The plates, especially when in masses, may be drawn out lengthwise by currents of fluid or by pressure. The remarkable viscidity of the plates can be demonstrated by placing a bit of filter paper at the edge of the cover-glass and thus causing currents in the fluid which fail to draw along even the isolated plates.

In order to see the regular and characteristic appearance of the fresh plates when arranged in masses, it is necessary that they should not be subjected to any pressure. When masses of plates are compressed even by the weight of the cover-glass they often appear to be made up wholly or in part of pale lines instead of coarse granules. This appearance of lines or threads seems to be due to an elongation and coalescence of the plates. These lines are often arranged with considerable regularity. They might be mistaken for threads of fibrin. A similar appearance of threads produced by coalescence of the plates is sometimes seen in hardened specimens where the threads are often varicose. Whether this appearance is due to the action of the hardening agent or to some other influence, such as the force of the circulating blood, must be left unsettled.

Although the plates make up the great mass of the thrombus leucocytes are present even at this early stage (during the first five minutes), and rapidly increase in number, so that at the end of half an hour the thrombus usually contains them in abundance. My investigations have not led me to assign so insignificant a rôle to the leucocytes in experimental thrombi as is done by Eberth and Schimmelbusch. I agree with

these authors in finding that thrombi produced mechanically in the manner mentioned consist in their inception essentially of blood plates. At the end of five minutes the leucocytes may be so scanty as not to attract attention. Usually, however, by this time clumps of leucocytes as well as scattered leucocytes are present here and there in the thrombus, and, as already mentioned, their number continues to increase. I have found them at the end of six hours, in mechanical thrombi, as numerous as in many human marantic thrombi. It is true that much diversity exists as regards the number of leucocytes even in thrombi of the same age, still it is the rule that white corpuscles, while they do not collect so rapidly or in such number as the blood plates, do accumulate and form a constituent part of experimental mechanical thrombi. In order to study the situation of the leucocytes sections of hardened specimens are necessary, but portions of fresh thrombi teased apart and treated with dilute acetic acid are favorable for ascertaining their number.

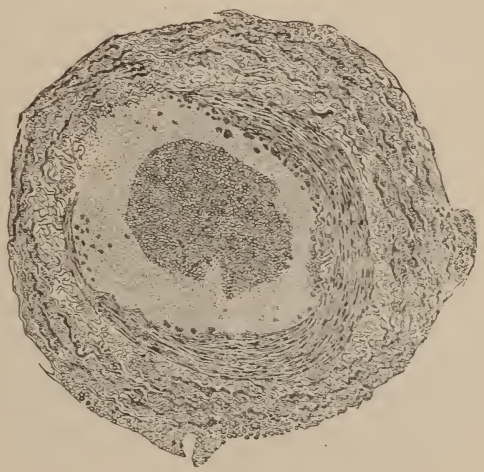
In searching for fibrin in fresh thrombi, it is important not to mistake for fibrin the threads produced by compression of the clumps of plates in the manner already mentioned. If these flattened-out masses of plates be washed with water, or, better still, with dilute Lugol's solution, there may be produced an appearance of interlacing and of parallel threads, which bear considerable resemblance to fibrin, but which are paler and which do not project beyond the margins of the clumps. Unmistakable fibrin in the form of fibrils, however, is to be found in experimental mechanical thrombi, but, so far as my observations go, not in the earliest stage of their formation. I have found fibrin in thrombi at the end of five minutes; frequently at the end of fifteen minutes, and usually at the end of half an hour. Fibrin is often found in so much larger amount in the fresh thrombi than in sections of the hardened thrombi that it is probable that it is formed in part after the removal of the vessel. As will be mentioned subsequently, fibrin can be demonstrated, also, in the hardened specimens, although not in the youngest thrombi. I am not able, therefore, to agree with Eberth and Schimmelbusch in denying altogether the presence of fibrillated fibrin in experimental thrombi produced by mechanical injury of the vessel, although our observations are in accord regarding the youngest thrombi.

I have dwelt thus at length upon the appearances of the fresh experimental thrombi because I have been unable to find any description of these appearances. With the exception of a brief allusion by Lubnitzky to sections of frozen thrombi, all the previous observations seem to have been made either upon the mode of formation of these thrombi in the living circulation or upon sections of hardened specimens. As has been

suggested by Weigert, and as is apparent from the foregoing description, a knowledge of the gross appearances of the plate thrombi is important in forming a judgment as to their nature. Sections are, of course, necessary to enable us to study more carefully the constituents of the thrombi and particularly their arrangement.

As already mentioned, I have made use chiefly of corrosive sublimate as a hardening agent, of paraffine as an imbedding medium, and of hæmatoxylin and eosin as staining agents.[1] In sections prepared in this way the plates can be seen with a distinctness and uniformity in shape that leave nothing to be desired. I am led to believe that most of the appearances which have been described as changes in the plates occurring during the first half hour (Lubnitzky and others), are due to imperfect methods of hardening. Eberth and Schimmelbusch recognize this fact in their preparations.

FIG. 1.



Section of dog's femoral artery containing thrombus formed in four minutes after forcible ligation of the vessel. Zeiss A. ocular 1.

Plate thrombi can be recognized in sections as well as in the fresh state by their peculiar translucence. I can only confirm the statements of Eberth and Schimmelbusch as to the composition of the experimental thrombi in their earliest formation. They are made up of blood plates. To the torn and partly detached internal elastic lamella as well as to the

[1] Since the delivery of this address I have also made use of Weigert's new method of staining fibrin on specimens hardened in alcohol.

lacerated media masses of plates are attached, which extend into the lumen of the vessel. Upon sections clumps of plates often appear to lie free in the lumen surrounded by blood, but subsequent sections show the connection of these clumps with others or with parts of the vascular wall. The thrombus often forms a complete ring around the torn inner wall of the vessel (Fig. 1). Not every injured part of the internal wall of the vessel is covered with a thrombus. One is often surprised to find such parts, even when severely lacerated, entirely free from thrombi or with only a thin layer of plates, recognized with a high magnifying power. The mass of plates composing the thrombus does not always appear uniform, but often, especially in older thrombi, there are lighter and darker portions, due apparently to greater density in the number of plates in some places than in others.

An appearance mentioned by Hanau is of interest, namely, the presence of a dark band around the margin of masses or islands of plates. Similar dense lines can often be seen running irregularly through sections of the thrombus. These denser bands have been interpreted as hyaline or as fibrin. It is not easy to determine their exact nature. They look like fibrin in many cases, but it is possible that they are produced by coalescence of the plates as the result of pressure or traction from the circulating blood in a manner similar to the lines which can be artifically produced in masses of fresh plates by pressure in the manner already described. Plates are not confined to the interior of the vessel, but when the internal elastic lamella and the media have been ruptured they often find their way in masses into the layers of the torn media or even into the adventitia. It is interesting to note the absence of any transition, as a rule, between the thrombus and the blood. The plates are just as dense usually at the margin of the thrombus as in the interior, and immediately adjacent to the peripheral plates come the red blood-corpuscles where the blood was still circulating before the vessel was removed.

Leucocytes are not generally present in large number in thrombi during the first five minutes of their formation. If serial sections be examined, here and there clumps of white corpuscles can be found even at this early stage. There are often more white corpuscles mingled with the plate masses in the coats of the vessel than in the thrombus proper. Leucocytes, scanty at first, continue to accumulate in larger and larger number, until they form a prominent part of the thrombus. I have found them in great abundance at the end of half an hour, although sometimes at this period their number is small. The leucocytes are generally arranged in clumps between masses of plates, although some

are usually scattered in among the plates. It is probable that the clumps of leucocytes are deposited in that form directly from the circulating blood. There is reason to believe that the leucocytes may also wander into the thrombus, for in sublimate specimens elongated nuclei, such as are seen in undoubtedly wandering white corpuscles in the wall of the vessel, can also be occasionally detected in the masses of plates. Moreover, the number of leucocytes within these masses increases with time. In successfully prepared sections the protoplasm can be seen around the nuclei of the white corpuscles, so that I do not agree with Lubnitzky that this has become merged with the plates. Sometimes the leucocytes are surrounded with a clear zone as if they lay in little spaces within the mass of plates, but this appearance is probably due to the action of the hardening fluid. Both uninuclear and multinuclear white corpuscles are present, but the latter predominate, and in the later stages many of the nuclei often appear much broken up.

Fig. 2.

From section of half-hour thrombus produced by forcible ligature of femoral artery of dog (see text). Shows masses of blood plates containing bands of fibrin and leucocytes. Zeiss D. ocular 3.

Although I have not seen any appearances which indicate that the white corpuscles disintegrate into granules, still non-nucleated white corpuscles can sometimes be detected, so that a necrosis or death of these corpuscles may take place within the thrombus. This does not seem, however, to be a common or extensive process.

As has already been stated, fibrillated fibrin is present in experi-

mental thrombi produced by mechanical injury of the vessel. It is not, however, found in the youngest thrombi, and the date of its appearance varies in different cases. I have found it in hardened specimens at the end of five minutes, but this is exceptional. It is not uncommon to find it at the end of fifteen minutes. I exhibit under the microscope sections of a thrombus of one-half hour's duration, in which there is a considerable amount of distinct fibrillated fibrin (Fig. 2). The amount of fibrin increases with the age of the thrombus, and in thrombi of twenty-four hours duration fibrin makes up usually a large part of the thrombus.

The fibrin appears in islands and bands between the masses of plates, and often extends in coarse fibres into the surrounding blood. The network is usually coarse, but fine threads are also present. After a time the clumps of white and of red corpuscles included in the thrombus are pervaded by a network of fibrin, whereas, this is absent in the dense clumps of plates. I have the impression that there is, in general, a relation between the number of leucocytes and the amount of fibrin, although the former appear in considerable quantity before the latter.

Inasmuch as in older thrombi (twenty-four to forty-eight hours) fibrin and leucocytes compose a large part of the thrombus, whereas, at its inception the thrombus is made up almost entirely of blood-plates, one is tempted to believe that the plates may be transformed into fibrin, but of this transformation I can bring no positive proof. The plate-masses, after a time, lose their regular granular appearance and appear darker in color and more homogeneous, but typical plates may be found in large number in thrombi forty-eight hours old.

It is apparent from the foregoing description, that experimental thrombi acquire with time all of the characteristics of human thrombi. The suspicion which has been raised that they represent a distinct class of thrombi, from the study of which we can draw no conclusion as to the formation of human thrombi, is unjustifiable. It is another question whether we are to suppose that all human white thrombi are formed in the manner described. Although I have not succeeded in producing permanent leucocytic thrombi experimentally, still there is every reason to believe that some human thrombi are composed from the beginning essentially of leucocytes. In observations which I have made recently for another purpose, of the living circulation in the mesentery of dogs, I have observed the formation of small parietal thrombi composed of white corpuscles, but these have invariably been washed away after a short time.

We may, it seems to me, think of the mode of formation of the experimental thrombi, which we have studied, and doubtless also of many human thrombi as follows: Given suitable conditions, such as alteration

of the vessel wall, slowing and irregularity of the circulation, the first constituents of the thrombus to accumulate are the blood plates. But although the plates collect at first in larger number and more rapidly, the leucocytes do not long remain absent, and in the course of time they are present in such quantity that they must be considered an essential constituent of the completed thrombus. At first the conditions for the coagulation of fibrin are not present, but with the increasing accumulation of leucocytes these conditions appear and fibrillated fibrin is deposited. It is in harmony with the current ideas concerning the cause of the coagulation of fibrin, to suppose that at first the fibrin ferment is absent, and that this is subsequently furnished by the leucocytes. The absence of fibrin in the early thrombi composed wholly of plates, is an argument additional to the evidence brought forward by Löwit and others, that the plates do not furnish the fibrin ferment. It is apparently only after the leucocytes have been included for a time in the thrombus that they die or undergo some alteration in their constitution which leads to the formation of the fibrin ferment. The final result is a plug composed of plates, leucocytes, and fibrin, and included red blood-corpuscles.

It seems to me an error to base our conception of the nature of a thrombus exclusively upon the constitution of the thrombus in its inception. While admitting that the thrombus is at first composed wholly of blood plates, we do not, as a matter of fact, meet with human thrombi in this early stage, or at least, only under exceptional circumstances. Our ideas as to the constitution of thrombi are based upon the examinations of the completed plugs which contain fibrin and leucocytes as well as plates. The study of the experimental thrombi enables us to form a clearer conception of the mode of formation of the thrombus, but does not necessitate any radical change in our ideas as to what constitutes a thrombus.

The question as to whether a thrombus is a coagulum or not, is, of course, open to discussion only regarding the plate thrombi in their earliest formation. Whether or not we are to regard the plate thrombi before fibrin has made its appearance as coagula, is a question which is not likely to be settled until we acquire more definite information as to the origin and nature of the blood plates. There is nothing in the gross appearances of these plate thrombi which would prevent us from considering them as soft, gelatinous coagula. Wooldridge, Löwit, and others believe that the plates are allied to fibrin but are not identical with it. I purposely avoid entering into any discussion here as to their existence in the normal circulation, for this is a point which must still

be regarded as *sub judice*, and which is not likely to be settled by the experimental study of thrombi.

The attempt of Eberth and Schimmelbusch to draw a sharp distinction between thrombi formed by conglutination and thrombi formed by coagulation, seems to me unwarranted. In the first place the process which they designate as conglutination may be, so far as we at present know, a form of coagulation. In the second place, whatever we may think as to the nature of the process of conglutination, the preceding investigations have demonstrated the transformation of conglutination thrombi into undoubted coagulation thrombi.

As regards the relation between changes in the walls of the vessels and thrombosis, I have reached the same conclusion as that expressed by von Recklinghausen, Eberth and Schimmelbusch, and others, that Cohnheim's views on this point were too exclusive. It is possible to produce experimentally severe injury of the internal coats of bloodvessels without any resulting thrombus. Among many positive results I have also in my notes the records of not a few negative results which have followed injury of the walls of the vessels by caustics, by forcible application of rough clamps, by scraping the interior of the vessel, etc. As is urged by these writers as well as by Weigert and others, slowing of the circulation and irregularities of the circulation produced by abnormalities in the lumen of the bloodvessels, are factors no less important in the production of thrombi than alterations in the vessel walls.

There is much which speaks for the correctness of the view advocated by Köhler, Hanau, and others, that some thrombi are caused by fermentative changes in the blood. Cases such as the one already mentioned, of extensive thrombosis of a large number of the bloodvessels throughout the body, are most naturally interpreted as examples of fermentation thrombosis. *April* 28, 1887.

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12. *Myxoma of neck of fœtus, causing hydramnion.*

Exhibited by Dr. D. Longaker.

This specimen was exhibited at the last meeting of the Obstetrical Society. It is of unusual interest, in that it illustrates the pathology of hydramnion in one class of cases, that in which the morbid conditions producing this form of dropsy are found in the fœtus.

The clinical history, in brief, is as follows: One week after the cessa-

tion of fœtal movements the patient miscarried. In three weeks she had suffered greatly from a rapidly increasing development of the abdominal tumor. No specific history.

The amount of amniotic fluid was over a gallon.

The fœtus is a male, of twenty-six weeks' development, weighs 1500 grammes, and measures 34 centimetres in length. Epidermis macerated.

Extending from mastoid to mastoid, and encroaching closely on the lower maxilla, there is a fluctuating tumor, covered with the thin integument. It extends down to the xiphoid, when the head is held between flexion and extension. The lower maxilla is imperfectly ossified, the symphysis still movable.

The head is markedly œdematous, especially the frontal region, the eyes being closed.

The peritoneum, pericardium, and pleura all moderately distended with serum, having a faint red color.

The heart, compared with that of another fœtus of corresponding period of development, shows a marked hypertrophy, being fully twice the normal size.

This appears to include both ventricles. The large vessels show a corresponding dilatation.

Nothing abnormal in thymus or in the lungs. Thyroid gland has not been discovered.

Kidneys not hypertrophied.

Suprarenal capsules normal.

No abnormality in any of the abdominal organs.

Umbilical cord œdematus.

Placenta, unfortunately, could not be examined.

Dr. Haehnlen, who studied sections from various parts of the tumor, reports that it is a myxoma.

Hydramnion resulted from increased pressure in the umbilical vein, the transudation into the amniotic cavity occurring through the capillary network situated beneath that portion of the amnion which covers the placenta, the so-called network of Jungbluth, the "chorionic boundary membrane," the vasa propria. This network is connected with the vessels of the umbilical cord, and the stasis existing here, in consequence of the same condition in the umbilical vein, explains the large and rapid increase of fluid in a very satisfactery manner.

I am enabled to exhibit this specimen through the kindness of Dr. Santee, with whom I saw the case. *May* 12, 1887.

13. *Intestine and sac from a case of strangulated femoral hernia.*

Exhibited by Dr. H. R. Wharton.

The patient from whom these specimens were removed, a woman of 50 years of age, was admitted to the University Hospital on February 8th with a tumor in the right groin, which she said had existed for some time, but had on the previous night become much larger and painful.

When admitted to the hospital she presented the symptoms of strangulated hernia. After failing to reduce the tumor by taxis she was etherized, and Prof. Ashhurst, under whose care she was, exposed the tumor and found that it consisted of a large mass of omentum and about twelve inches of deeply congested gut. The seat of constriction was found to be at the neck of the sac, and as after dividing this the gut improved in color, it was reduced. The omentum was found to be irreducible and was therefore ligated and cut off, leaving the stump in the wound. On the day after the operation the patient exhibited signs of collapse and died about thirty-six hours after the operation. A post-mortem examination was made, and a portion of the congested gut was found engaged in the neck of the sac; there was no evidence of peritonitis, and death appeared to have resulted from collapse due to the recurrent strangulation of the gut, and probably also to beginning gangrene.

February 10, 1887.

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14. *Two cases of four leaflets to the pulmonary valve.*

Exhibited by Dr. W. Osler.

These two cases represent a not very uncommon anomaly of the pulmonary valve, in which a small space left between adjacent cusps is occupied by a fourth segment. The cases presented no points of clinical interest bearing upon the heart.

Case. I.—Between two cusps there is a space 3–7 mm. in width, which is occupied by a small semilunar valve. It has a well-marked curvilinear base of attachment, the free margin is 8 mm. in length, presents no corpus Arantii, and is anchored to the artery wall by a small narrow tag. The depth of the valve is 8 mm. The lateral attachments are to the adjacent cusps, not directly to the artery. Two or three fenestra-

tions of the large cusps communicate directly with the pouch of the small one. The three segments are of equal size, and normal.

Case II.—Between two normal looking segments there is placed a small cusp 7 mm. in width, 8 mm. in depth, with a distinct crescentic margin of attachment and a curved free border. The lateral edges are united to the contiguous valve. The sinus is distinct and does not communicate with the adjacent ones. The adjacent cusps look a little smaller than the third. *November* 11, 1886.

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15. *Bicuspid pulmonary valve.*

Exhibited by Dr. W. Osler.

In a case of carcinoma ventriculi, with enormous secondary mediastinal growth, the pulmonary valve presented the following peculiarities. The orifice was guarded by two segments. The smaller measured 33 mm. along the free border, was 15 mm. across the face, presented two fenestrations, and there was no distinct corpus Arantii. The other segment measured 36 mm. and across the face 15 mm. The free border was thickened, presented no distinct corpus Arantii. The body of the leaflet was also a little thickened. At the attached margin there was a slight indication of separation into two segments. On the arterial face there was a small median raphe which passed from the arterial wall to the base of the segment. Here it expanded into a series of radiating fibres which extended along the inner surface. This median raphe separated two sinuses of about equal size. *December* 9, 1886.

XI. CARD SPECIMENS.

a. The digestive apparatus.

1. Congenital cystic liver. J. M. Keating. December 10, 1885.
2. Congenital diaphragmatic hernia. H. F. Formad. Dec. 10, 1885.
3. Congenital occlusion of the rectum. H. F. Formad. December 10, 1885.
4. Fatty liver, weighing ten pounds. T. S. K. Morton. Jan. 14, 1886.
5. Series of specimens of gastric ulcer. H. F. Formad. February 25, 1886.
6. Angioma of liver. H. F. Formad. February 25, 1886.
7. Traumatic rupture of liver and stomach. H. F. Formad. March 11, 1886.
8. Gastric ulcer. F. X. Dercum. March 11, 1886.
9. Stomachs from cases of carbolic acid, sulphuric acid, and arsenic poisoning. H. F. Formad. June 10, 1886.
10. Umbilical hernia. H. F. Formad. June 10, 1886.
11. Diverticulum from the ileum. H. F. Formad. June 10, 1886.
12. Some very large and very small gall-bladders. H. F. Formad. June 10, 1886.
13. Tonsillitis. H. F. Formad. June 10, 1886.
14. Red atrophy of the liver. H. F. Formad. June 10, 1886.
15. Hydatid cyst of the liver. J. H. Musser. September 9, 1886.
16. Calculi from the right iliac fossa. J. H. Musser. September 9, 1886.
17. Tubercular peritonitis. J. H. Musser. September 23, 1886.
18. Intestinal cast. J. P. C. Griffith. September 23, 1886.
19. Mucous disease. J. K. Young. November 11, 1886.
20. Intestine passed by the anus. W. Osler. December 9, 1886.
21. Cancer of the colon. W. Osler. December 9, 1886.
22. Mucous colitis, with membrane *in situ*. W. A. Edwards. December 23, 1886.
23. Enormous ulcer of the stomach from a case of melancholia. F. X. Dercum. December 23, 1886.

24. Cancer of the stomach from a case of melancholia. F. X. Dercum. December 23, 1886.
25. Cirrhosis of the liver. H. M. Fisher. December 23, 1886.
26. Ulcer of the stomach. H. M. Fisher. January 13, 1887.
27. Floating liver. H. F. Formad. January 27, 1887.
28. Three cases of biliary calculi. T. D. Dunn. January 10, 1887.
29. Cirrhosis of the liver. H. F. Formad. March 10, 1887.
30. Liver, with very large gall-bladder. H. F. Formad. March 10, 1887.
31. Hemorrhagic infarct of the stomach. H. F. Formad. March 10, 1887.
32. Abscess of the gall-bladder from biliary calculi. M. O'Hara. March 24, 1887.
33. Syphilitic liver. F. P. Henry. March 12, 1887.
34. Duodenal ulcer. J. P. C. Griffith. June 23, 1887.

b. The vascular system.

1. Dissecting aneurism of the root of the aorta. H. F. Formad. November 12, 1885.
2. Acute pericarditis. J. Tyson. December 10, 1885.
3. Tricuspid insufficiency and right-sided hypertrophy of the heart. R. G. Curtin. December 10, 1885.
4. Chronic endo- and peri-carditis; heart weighing fifty-six ounces. J. S. Neff. December 10, 1885.
5. Acute pericarditis. H. F. Formad. December 10, 1885.
6. Heart with three ventricles. H. F. Formad. December 10, 1885.
7. Series of cases of dissecting aneurism of the root of the aorta. H. F. Formad. February 25, 1886.
8. Embolic myocarditis. H. F. Formad. February 25, 1886.
9. Hypertrophied heart; weight forty ounces. H. F. Formad. March 11, 1886.
10. Heart with aortic disease, from a case of mania. F. X. Dercum. March 11, 1886.
11. Aneurism of the renal artery, the result of embolism. W. Osler. May 13, 1886.
12. Aneurism of the aorta. H. F. Formad. June 10, 1886.
13. Atheroma of the aorta. H. F. Formad. June 10, 1886.
14. Dissecting aneurism of the aorta. H. F. Formad. June 10, 1886.
15. Two cases of organic heart disease. J. H. Musser. November 11, 1886.

16. Acute pericarditis. H. F. Formad. December 23, 1886.
17. Acute pericarditis. H. M. Fisher. January 13, 1887.
18. Sudden death from aortic insufficiency. H. F. Formad. March 10, 1887.
19. Valvular heart disease. S. W. Morton. March 24, 1887.
20. Heart disease from a case of acute phthisis. F. P. Henry. May 12, 1887.
21. Dissecting aneurism of the aorta. H. F. Formad. May 12, 1887.
22. Valvular cardiac disease. H. F. Formad. May 12, 1887.
23. Aneurism of the aorta rupturing into the pleural cavity. H. F. Formad. June 9, 1887.

c. The organs of respiration.

1. Gunshot wound of the larynx. H. F. Formad. March 11, 1886.
2. Acute phthisis, with heart disease. H. F. Formad. June 10, 1886.
3. Acute phthisis. W. Osler. June 24, 1886.
4. Fibroid phthisis, with tuberculosis. J. H. Musser. November 27, 1886.
5. Two specimens of nasal polypi. C. Seiler. January 13, 1887.
6. Papilloma of the larynx. C. Seiler. January 13, 1887.
7. Lungs from a case of acute phthisis. H. F. Formad. January 27, 1887.
8. Tumor of the larynx. E. Martin. February 10, 1887.
9. Two cases of diphtheria of the larynx. J. P. C. Griffith. June 23, 1887.

d. The genito-urinary apparatus.

1 Fluid from a cyst connected with the spermatic cord. C. B. Nancrede. November 12, 1885.
2. Extra-uterine pregnancy. H. F. Formad. November 12, 1885.
3. Rhabdo-myoma of kidney. H. F. Formad. November 12, 1885.
4. Two cases of extra-uterine pregnancy. H. F. Formad. January 28, 1886.
5. Two uteri, with children at full term in position. H. F. Formad. January 28, 1886.
6. Myxomatous cyst of the ovary. H. F. Formad. January 28, 1886.
7. Cyst of the uterus. H. F. Formad. January 28, 1886.
8. Series of uteri from criminal abortions. H. F. Formad. January 28, 1886.

9. Pregnant uterus, death from purulent pericarditis. H. F. Formad. March 11, 1886.
10. Fibroids of uterus and ovaries, from a case of recurrent mania. F. X. Dercum. March 11, 1886.
11. Amyloid kidneys from a case of phthisical insanity. F. X. Dercum. March 11, 1886.
12. Undescended testicle. H. F. Formad. March 25, 1886.
13. Pregnant uterus. H. F. Formad. March 25, 1886.
14. Series of uteri from criminal abortions. H. F. Formad. April 8, 1886.
15. Extra-uterine pregnancy. H. F. Formad. May 13, 1886.
16. Diphtheritic cystitis. H. F. Formad. June 10, 1886.
17. Horseshoe kidney. J. H. Musser. September 9, 1886.
18. Sarcoma of the breast; death from hemorrhage. H. F. Formad. September 23, 1886.
19. Anomalous and congenitally dislocated kidneys from a case of dementia. F. X. Dercum. December 23, 1886.
20. Horseshoe kidney. J. H. Musser. January 13, 1887.
21. Urinary calculi. H. M. Fisher. January 13, 1887.
22. Cystic kidney. H. F. Formad. January 27, 1887.
23. Kidneys with calculi. H. F. Formad. January 27, 1887.
24. Uterus from a criminal abortion. H. F. Formad. January 27, 1887.
25. Sarcoma of the breast. H. F. Formad. January 27, 1887.
26. Recurrent tumor of the breast. E. Martin. February 10, 1887.
27. Uterus and appendages from a case of salpingitis in a child. D. Longaker. May 12, 1887.
28. Epithelioma of the penis. H. R. Wharton. June 26, 1887.
29. Slides of epithelioma of the penis. G. E. de Schweinitz. June 23, 1887.
30. Slides of cancer of the ovary. G. E. de Schweinitz. June 23, 1887.

e. The nervous system.

1. Falx cerebri, showing bony plate. T. S. K. Morton. December 10, 1885.
2. Two cases of spina bifida. H. F. Formad. December 10, 1885.
3. Brain tumor. G. Hinsdale. January 28, 1886.
4. Brain showing ossified falx. H. F. Formad. February 25, 1886.
5. Cerebro-spinal fever. F. Woodbury. March 25, 1886.
6. Hæmatoma duræ. F. X. Dercum. April 8, 1886.

7. Some rare forms of cerebral apoplexy. H. F. Formad. April 8, 1886.
8. Brain tumor. H. F. Formad. June 10, 1886.
9. Extra-dural hemorrhage from fracture. H. F. Formad. September 23, 1886.
10. Old hemiplegia. W. Osler. January 13, 1887.
11. Cerebral hemorrhage. H. F. Formad. March 13, 1887.

f. Specimens from the lower animals.

1. Cyst from the back of a cow. T. D. Dunn. September 10, 1885.
2. Cystic kidney from a calf. H. F. Formad. January 28, 1886.

g. Miscellaneous.

1. Tænia flavopunctata. W. Pepper. September 24, 1885.
2. Tænia mediocanellata. W. Pepper. September 24, 1885.
3. Head and neck of a tænia solium. W. A. Edwards. December 10, 1885.
4. Certain pseudo-parasites. W. Osler. February 11, 1886.
5. Strangled infant. H. F. Formad. March 11, 1886.
6. Specimens from a case of chloral poisoning. H. F. Formad. April 8, 1886.
7. Tubercular organs from a child. H. F. Formad. May 13, 1886.
8. Lipoma of the pelvis. H. F. Formad. September 23, 1886.
9. Post-sternal abscess. H. F. Formad. December 9, 1886.
10. Leg ulcer; death from hemorrhage. H. F. Formad. January 13, 1887.
11. Lesions of syphilis. H. F. Formad. January 13, 1887.
12. Stab-wound of the abdomen. H. F. Formad. April 26, 1887.

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